THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—15TH YEAR. SYDNEY, SATURDAY, JANUARY 21, 1928. No. 3.

Authors of articles submitted for publication are requested to read the following instructions and to comply with them.

All articles must be typed with double or treble spacing. Carbon copies should not be sent. Abbreviations should be avoided, especially those of a technical character at times employed in ward notes. Words and sentences should not be underlined or typed in capitals. The selection of the correct type is undertaken by the Editors. When illustrations are required, good photographic prints on glossy gaslight papers should be submitted. Each print should be enclosed in a sheet of paper. On this sheet of paper the number of the figure and the legend to appear below the print should be typed or legibly written. On no account should any mark be made on the back of the photographic print. If no good print is available, negatives may be submitted. Line drawings, graphs, charts and the like should be drawn on thick, white paper in India ink by a person accustomed to draw for reproduction. The drawings should be large and boldly executed and all figures, lettering and symbols should be of sufficient strength and size to remain clear after reduction. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available. The reproduction of all illustrations but especially of skiagrams entails the sacrifice of time and energy and is expensive. Authors are expected to take a corresponding amount of trouble on the preparation of their illustrations, whether skiagrams, photographs, wash drawings or line drawings. The references to articles and books quoted must be accurate and should be compiled according to the following scheme. The order should correspond to the order of appearance in the article. The initials and surnames of the authors, the full title of the article or book, the full (unabbreviated) title of the journal in which the article appears, the date of the issue (day, month and year) and the number of the first page should be given in this sequence.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.

"The Mechanism of Pain Production in Abdominal Visceral Disease with Special Reference to the Pains of Peptic Ulcer," by VICTOR J. KINSELLA, M.B., Ch.M. 64

REPORTS OF CASES—

"Acute Nicotine Poisoning," by F. A. E. LAWES, M.B., Ch.M. 84

ANALYTICAL DEPARTMENT—

"Caprokol" 84

LEADING ARTICLES—

A Retrospect 85

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—

Dermatology 90
Urology 90

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 92
Medico-Political 93
Nominations and Elections 94

MEDICAL SOCIETIES— PAGE

Medical Defence Society of Queensland 94

SPECIAL CORRESPONDENCE—

London Letter 94

CORRESPONDENCE—

The College of Surgeons of Australasia 95
Pathological Service 96

WILLIAM GIBSON RESEARCH SCHOLARSHIP FOR MEDICAL WOMEN 97

COMMERCIAL FLYING IN THE UNITED STATES 97

NAVAL AND MILITARY—

Appointments 97

BOOKS RECEIVED 98

DIARY FOR THE MONTH 98

MEDICAL APPOINTMENTS 98

MEDICAL APPOINTMENTS VACANT, ETC. 98

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 98

EDITORIAL NOTICES 98

THE MECHANISM OF PAIN PRODUCTION IN ABDOMINAL VISCERAL DISEASE WITH SPECIAL REFERENCE TO THE PAINS OF PEPTIC ULCER.

By VICTOR J. KINSELLA, M.B., Ch.M. (Sydney), *Melbourne.*

FOR ages man considered the viscera of his abdomen to be highly sensitive to pain and looked upon disembowelling as one of the most terrible deaths. Generations have stood horrified and perhaps sceptical before the picture of the death of Cato Uticensis, the austere old Roman who preferred evisceration by his own hand to capture by the legionaries of Cæsar. But old ideas have been modified in the crucible of experiment and in recent years the surgeon invading the abdomen has tried and found wanting time-honoured beliefs. The second stage of a colostomy, without anæsthetic, has shown that the bowel is insensitive to cutting and burning. Some of us have seen the intestines escape through the opening of a laparotomy wound; we have removed the dressing, manipulated the bowel without causing pain and wondered whether the death of Cato was so agonizing after all. In spite of this apparent absence of sensibility in the viscera we know that their disorder may produce pain of all kinds and degrees. Pain in visceral disturbance is as ancient as mankind and yet the pioneers who realized the difficulties in understanding it and first attempted to explain it, belong to but recent times. Much work and much thought have been expended on the problem in the last thirty years and still no completely satisfactory solution has been found. Observations have accumulated, some consistent, some contradictory and deductions have been made, some logical, some inadmissable. Before attempting an advance I propose to sift the evidence already before us and to determine what must be retained and what must be rejected.

THE INSENSIBILITY OF THE VISCERA TO ORDINARY STIMULI.

That the viscera can be burnt with cautery or caustic, cut, stitched, pinched and crushed without causing pain, was first pointed out by Mackenzie[1] and Lennander[2] and has now become a matter of common knowledge since laparotomy under local anæsthesia has taken its place in surgical procedure. It has, therefore, been concluded that the bowel is normally insensitive to these stimuli and, as far as I know, Kast and Meltzer[3] have been the only dissentients. They find that the intestines of dogs are very sensitive to pain and that they lose their sensitiveness after the injection of cocaine into a remote region. They remind us that the opening of the abdomen with the rapid escape and exposure to air of its contents inhibits the motor activity of the intestines and they argue, by analogy, that sensibility is likewise depressed. They conclude that the insensibility of the viscera in men is artificial and due probably to (*a*) the general effects of the local anæsthetic and (*b*) to the exposure.

With regard to the local anæsthetic one of Mackenzie's patients was operated upon without this. To investigate the question further J. F. Mitchell[4] took the rather heroic step of performing two operations (the first an exploratory laparotomy and the second the cure of a large inguinal hernia) after infiltrating the skin with normal saline solution. The patients complained bitterly as each layer of abdominal parieties was opened. But the viscera were insensitive. Again, we sometimes see the surgeon delay twenty-four hours or more in opening a segment of gut exposed for colostomy. By this time the injected drug has disappeared and yet the subject complains of no pain.

We can settle the question in man as regards both exposure and local anæsthesia by considering the Mickulicz operation for carcinoma of the colon. In this operation the affected segment of bowel is removed and an artificial opening is made, the afferent and efferent loops being left within the abdomen stitched together in double-barrelled gun fashion. I have seen two such patients in whom the spurs between the two loops were crushed by clamps three or four days after the primary operation. Pain was experienced by neither patient. There could be no question of anæsthesia by drugs or exposure under these circumstances.

For a possible explanation of the difficulties raised by these investigators we may turn to Carlson and Luckhardt[5] who found that stimulation of the viscera can produce defensive or escape types of skeletal reflexes, even in decerebrate and spinal preparations of the frog and turtle. Therefore, these reflexes do not of necessity indicate pain and yet it is upon these that animal experimenters depend to a large extent for their evidence of pain. Moreover, Müller and Hotz, quoted by Meeker,[6] repeated the experiments of Kast and Meltzer with opposite results. We can, therefore, safely conclude that the viscera in the human subject are insensitive to experimental stimuli which are painful when applied to other parts.

THE BIOLOGICAL SIGNIFICANCE OF PAIN.

This peculiar difference in the reaction of different tissues in man refers us back to the third week of embryonic life. The cells of the primitive embryonic mesoderm which had formed a single layer, separate into two layers, the parietal or somatic joining the ectoderm and the visceral or splanchnic joining the entoderm. Differentiation at such an early stage of development suggests a profound difference in biological significance.

The first of these layers, the somatopleure, will come into contact with the environment and will adjust the organism accordingly. It, therefore, contains the receptive or sensory mechanism which will acquaint the creature with the nature of its surroundings and it contains the locomotor system which will enable an advance or a retreat to be made, according to the requirements. The point of the knife, for example, can be appreciated and interpreted as a possibly harmful influence, a menace to the existence of the organism; hence the painful impression and the immediate response. The derivatives of this layer may be compared with the navigat-

ing bridge and the engine room of a ship; an officer keeps watch, ready to signal the necessary alterations in course or in speed and the engines are altered accordingly.

The second layer, the splanchnopleure, becomes in time completely wrapped up by the first layer; it has no opportunity of contact with the outside world and is concerned only with the internal workings of the animal. It has no concern in the search for or capture of food, but is responsible for its disposal, once caught. It has no concern with the perception of and flight from danger, but it is responsible for supplying the rest of the body with fuel and oxygen to enable necessary adaptations to be made. Its derivatives may be compared with the stokehold and furnaces of the ship; it cannot receive and interpret and react to impressions from the outside world; it has no anatomical opportunity of so doing, neither has it any need of such a function which is exercised in so efficient a manner by its brother layer, the somatopleure.

The latter layer is equipped with a highly complicated mechanism, especially designed for making the appropriate adjustments to environmental influences. It helps to form special-sense organs, its epithelial tissues are supplied by the delicate ramifying fibrillæ of sensory nerves and its connective tissues with the special end-organs called after Krause and Pacini and others; its effector apparatus is muscle tissue characterized by rapid response and the whole is presided over by a central directing station, the brain, in which the impulses travel along clearly defined uni-directional paths. Accurate localization and rapid, purposeful reaction are the physiological result. The intestines, on the other hand, are poorly supplied with special sensory end-organs, their muscular tissue responds sluggishly and their nervous system is of the primitive net type in which there is no central station comparable to the brain. The parts are to a certain extent autonomous and impulses are transmitted in comparatively diffuse fashion throughout the nerve-net. Physiologists have long remarked that our splanchnic nervous system is comparable to the lower invertebrate system as seen in the sea anemone. In reality it is less complete from a biological point of view, on account of the division of function which has taken place in our development. Is it any wonder then that the gross stimuli of environmental influences, such as cutting and burning are not perceived by the visceral layer? The question then arises, what is the mechanism which produces the pain in visceral disturbance, what is the "adequate stimulus"? Pain, as we have seen, is a defensive reaction of the organism to conditions which menace its safety. It cannot be produced in our laboratories by subjecting the splanchnopleure to those conditions which produce somatic pain, for these conditions have nothing whatever to do with our viscera in the course of nature. We must look to disease if we are to find the adequate stimulus, because unmistakable pain is produced in Nature's visceral experiments.

I shall now review the finding of some of those who have sought the solution of the problem. Although the work done by each is of great importance, it will be seen that each goes but a part of the way, leaving much still to be explained.

Sir James Mackenzie's Work.

Mackenzie[1] taught that stimuli from diseased viscera cause no sensation themselves, but that they cause an irritable focus in the spinal cord and that normal impulses from the muscles and skin reaching this focus give rise to the painful impressions which are referred to the parietes. Between neurones there is a definite threshold of resistance to the passage of nervous impulses and this serves to keep the impulses to definite paths required for the normal working of the organs. But the extraordinary stimuli set up in the viscera by disease are transmitted by the sympathetic system to the spinal cord and may break down the synaptic barriers and overflow to neighbouring somatic neurones, thus producing pain and other reflex effects. This overflow of nervous impulses to neighbouring centres in the central nervous system explains much and we are indebted to Mackenzie for a great advance in our understanding of symptoms. We can now comprehend the peculiar distribution of pain far removed from the affected viscus, for example, the shoulder pain in gall-bladder disease and the radiating pain of renal colic. We can understand the phenomena sometimes associated with visceral pain, the hyperalgesia of the skin and muscles, the frequent micturition sometimes noticed in appendicitis and the vomiting and pallor which accompany severe colic. It is strange that Mackenzie did not include the dyspeptic or gastric manifestations of gall-bladder and appendical disease among his reflex symptoms, an overflow not only to somatic, but to other splanchnic centres in the cord.

At this point I shall digress for a moment to consider the interesting question asked by Head:[7] "Why is pain referred?" He compares referred pain with allocheiria. Allocheiria is the mistaken localization of a stimulus applied to an area of skin, the sensibility of which has been diminished by a disease such as *tabes dorsalis* and if this area, for example on the leg, is pricked by a pin, the pain is localized in a more sensitive part, perhaps on the opposite limb. And so a painful stimulus of the internal organs is conducted to the cord and brought into association with the fibres for painful sensation from the surface of the body. "But the sensory and localizing power of the surface of the body is enormously in excess of that of the viscera and thus by what may be called a psychical error of judgement . . . the pain is referred on to the surface of the body, instead of on to the organ actually affected." This comparison is very interesting, although it does not really supply the answer to the question. The answer is to be found rather in a consideration of the defensive function of pain.

So far we can follow Mackenzie with advantage but, as we shall see, his ideas fall short of the whole story in two important respects.

He demonstrated the insensibility of the viscera to certain forms of stimuli and concluded that pain and tenderness are felt not in these structures, but

only in the peripheral tissues. He neglected the possibility of pain production by stimuli other than those employed by himself and of pain production in disease by those stimuli which produced no pain in health. Indeed, there is abundant evidence that, if we could get beneath the abdominal wall and press upon the diseased organ without disturbing the parietes, pain would result. In clinical examination tenderness is daily demonstrated in diseased organs, sometimes in striking manner when they descend on inspiration against the examining hand. Since the organs have been directly visualized and palpated under the fluoroscopic screen, definite tenderness has been proved in the ulcer niche, in the diseased appendix and lately in the diseased gall-bladder.

The insensibility of the healthy viscus or of the viscus in a painless stage of disease has been proved time and again by operations under local anæsthesia, but scant attention has been paid to the equally or more important conditions in which the viscus is the site of a lesion painful at the time of operation. Unfortunately, we rarely have the opportunity of observing a laparotomy under these conditions, because even those surgeons who champion local anæsthesia, for example Labat, advise against its use in acute abdominal emergencies and, while they are quite willing to use the method in the "interval operation" for appendicitis, that is in the painless stage, they will not as a rule use it when the patient is in the painful acute stage. However, Professor Finsterer[8] finds in resecting the stomach for ulcer or cancer that anæsthesia of the abdominal wall is adequate until the abdomen is opened and then pain is produced by handling the diseased viscus. This can be prevented by mesenteric anæsthesia which is "entirely satisfactory in cases of resection for a movable ulcer or cancer in a ptotic stomach," that is in a case where dragging can be avoided, but when this is impossible, the peritoneum of the posterior abdominal wall must be anæsthetized.

The sensibility of our organs is suggested by the fact familiar to most of us, that pain, for example, of intestinal colic, often appears to be deeply situated and the sufferer has no doubt that it is coming from within the abdomen. Then again how can the unilateral symptoms and signs of appendical and gall-bladder disease be explained unless by the sensibility of the viscera themselves? Mackenzie himself admitted the inadequacy of his theory to explain these pains which should be in the mid-line, according to his views, for the appendix and gall-bladder are developmentally part of the digestive tube and share in its nervous connexion.

The weight of evidence proves that there is a truly visceral as well as a referred component in abdominal pain.

The second shortcoming in Mackenzie's teaching is the little help which he gives us in explaining the ultimate visceral condition which produces pain. His referred pain depends upon an irritable focus in the spinal cord which merely shifts the problem without solving it. The visceral stimulus which produces this irritable focus, is still to be found. Mackenzie does not concern himself so much with the nature of the adequate stimulus as with its effects and their reflex paths. He goes part of the way, however, and observes that if a distending enema is given and retained, pains result. That these are due to peristalsis "is evident from the fact that with the relaxation of the sphincter during the pain, the contents of the bowel are expelled with considerable force and the pain at the same time subsides." Then he describes the operation referred to above, in which peristalsis synchronized with the pains, and he comes to the conclusion that severe pain can be elicited by the violent contractions of hollow muscular organs. Some inkling of this truth may have first presented itself to man at the birth of Cain. At any rate the observation is a very old one and pain has long been associated with violent activity of the viscera, for example in intestinal, renal and biliary colic. Beaumont[9] in 1833 noted the pains which accompanied spasmodic contractions of the pylorus upon the thermometer introduced into the stomach of St. Martin. More recent work, notably by Carlson, has confirmed these observations. I am dealing now with the disturbance of the law which requires a wave of inhibition to precede the wave of contraction in normal peristalsis, the "law of the intestine" (Bayliss and Starling) or the "law of contrary innervation" (Meltzer).

It may be accepted as truth that violent contractions of hollow muscular organs can produce pain, but many observers have been led upon false trails through overlooking the fundamental fact that pains known to depend on such a stimulus, have definite and characteristic attributes grouped under the term "colicky." Consider the pains of labour and the colic of renal biliary and intestinal affections. They are essentially paroxysmal. The paroxysms may be separated by intervals quite free from pain or they may merge and produce a wave-like effect, alternately waxing and waning. The onset is comparatively abrupt and the maximum degree of pain is reached after a few seconds and often the pains radiate over extensive areas, perhaps even into the limbs. Having grasped this principle, I shall not attempt to explain by violent contractions of hollow muscular organs, pains which have not these attributes.

But let it be noted that associated with these paroxysmal pains there is sometimes pain of a different variety and of a different mechanism with which I shall deal later.

Lennander's Hypothesis.

Lennander,[2] like Mackenzie, realized the insensibility of the viscera and his fellow-countryman Ramström, Professor of Anatomy at Upsala, showed that the subserous layer of abdominal wall is richly supplied by cerebro-spinal nerves. They noted the sensitivity of this layer and that pain is produced by handling the viscera if the parietal attachments are dragged upon. Their experiments showed that the parietal peritoneum of the anterior abdominal wall is like the cornea and possesses only a sense of pain, not pressure, cold or heat. So far, all is well, but

they concluded that "all painful sensations within the abdominal cavity are transmitted only by means of the parietal peritoneum and its subserous layer." Such a sweeping theory, of course, has many difficulties to face and its sponsors were forced to many unfounded speculations. For example, to explain the pain in infectious processes they postulated a spread of lymphangitis and lymphadenitis from the mesenteries along the lymphatic vessels to the subserous tissue of the abdominal wall. When the pain in these conditions is first diffuse and later localized, for example in acute appendicitis, they imagine a lymphangitis or peritonitis of a large portion of the parietal peritoneum which soon passes off except in a restricted area. Colicky pains are ascribed "partly to a stretching of the parietal attachments of the bowel and partly to a displacement of the serous lining of the abdominal wall." To show these pains experimentally a piece of ileum forty centimetres long was clamped and inflated with air; "the bowel straightened, the mesentery got stiff and assumed a fan-shaped form" and stood out from the vertebral column and the patient complained of pain which passed off when the mesentery resumed its normal position. No doubt this pain was due to dragging on the mesentery, but can such conditions of the bowel and mesentery be assumed to occur under ordinary conditions? Then let us consider renal and biliary colic. The difference in excursion of the muscular walls of the respective ducts in normal and in colicky contractions cannot be more than a few millimetres. How can these few millimetres make such a difference to the parietal peritoneum that no sensation is produced in one case and agonizing pain in the other? Greater disturbance is produced by every normal peristaltic wave in the pylorus and duodenum and yet no pain results, even when there is an associated lymphangitis, for example in that pain-free period after a meal in peptic ulcer. Again, how are we to explain, according to Lennander's hypothesis, the relief of peptic ulcer pains by alkalis? We shall have to suppose that alkalis arrest digestive peristalsis, but it will be shown later they do nothing of the kind.

Lennander's conjectures have carried him farther than his observations warrant, but still we learn from him the important facts that the parietal peritoneum is very sensitive and that pain is produced by dragging on it. Thus, in Rubin's test for the patency of Fallopian tubes, gas collects under the diaphragm and causes pain by stretching the ligaments of the liver. The presence in the parietal peritoneum of the sensory corpuscles of Pacini is of significance. Its sensitivity plays a terrible part when it is suddenly bathed in a rush of some foreign irritating fluid, as in the rupture of a peptic ulcer. But of more usual occurrence is the dragging which produces pain in enteroptosis, nephroptosis and *prolapsus uteri*. In such cases the pain is of a "dragging" nature, aggravated by standing, walking or working or anything which increases the mechanical drag and relieved by support of the fallen organ. Again we find that pain depending on a definite mechanism has definite and characteristic attributes.

Hurst's Hypothesis—Distension.

Although Mackenzie and Lennander showed that the viscera are insensitive to cutting, pinching and so on, "it still remained possible," as Hurst remarked[11] [12] "that the viscera would be found sensitive were the adequate stimulus employed." He continued: "It might be expected that tension on the fibres of the muscular coat would prove to be the adequate stimulus" and concluded that "a certain degree of distension leads to the sensation of fullness, if the internal pressure is slowly raised; the same distension rapidly produced leads to the sensation of pain." He supported this contention by producing a sensation of fullness in the œsophagus, colon and rectum by introducing and gradually inflating a rubber balloon and in the stomach by gradual air-inflation. Pain is produced by rapid inflation of the balloon in the first three organs and by rapid inflation of the stomach with carbon dioxide produced by swallowing separately sodium bicarbonate and tartaric acid. Hurst came to the conclusion that "the only stimulus to visceral pain is distension." It is possible for such distension to take place in the natural course of events, but that this is the usual cause of abdominal pain appears to be unlikely, for the following reasons.

In regard to the healthy stomach, Dr. Apperly informs me that the rapid distension with air (250 cubic centimetres to 1,250 cubic centimetres) of the stomach of eight healthy students produced fullness, but no pain, the experiments having been interrupted as soon as any sensation was felt. Distension of my own stomach with water poured down a Rehfuss tube at a pressure head of ninety centimetres, resulted in fullness, then nausea, then vomiting, but no pain. Dr. Apperly and I swallowed water as rapidly as possible and with great difficulty succeeded in producing pain and tenderness high in the epigastrium, preceded by nausea and fullness. The pain was aggravated by movements of the trunk, especially flexion, and it therefore constrained us to remain stiff and erect during the few minutes of its persistence. It is natural that pain caused by distension should be aggravated by flexion of the trunk which tends to increase intra-abdominal pressure, but patients with colicky pains and sometimes even with ulcer pain may find a certain amount of relief in the flexed attitude, thereby suggesting that their pains are not due to distension.

With regard to the stomach in disease it is common experience to distend the organ, for example in gastric lavage or barium examinations, and observe the patient become uneasy and complain of fullness and later of nausea. Frequently they give warning that they will vomit, if the filling process is continued. These observations go to show that although distension of the stomach can produce pain, fullness and nausea and perhaps vomiting are present prior to and more constantly than the pain. Pain in stomach affections, for example peptic ulcer, is often unaccompanied by fullness, nausea

and vomiting. When present these symptoms have often no necessary association with the pain. Again it is important to note that the pain can be produced only if distension is carried to an heroic degree. Thus, it required a very determined effort on Dr. Apperly's and on my part to overcome the fullness and nausea first produced and to carry the experiment to pain production. In the inflation experiments quoted by Hurst, 1,700 cubic centimetres of carbon dioxide were suddenly formed in the stomach of normal individuals, who then complained of an unpleasant feeling and "often" (but not always) "a painful sense of fullness which is relieved by eructation." To my mind these experiments demonstrate, not the natural adequate stimulus, but rather the difficulty of producing pain by distension of the stomach. Pain should be produced readily, as when the renal pelvis is filled for pyelography, if distension be a natural, adequate stimulus for pain production. Moreover, we shall see that the pain of gastric ulcer occurs usually in the normally active organ and often in the hypotonic (or long) stomach of poor peristaltic activity.

The administration of the opaque clysma for radiographic purposes has proved most interesting and instructive. I make a practice of filling the colon gently with the container one foot above the patient, until the fluid has reached the caecum. No pain, usually not even discomfort, results and the colon remains motionless. But occasionally I raise the container three or four feet above the patient; the colon becomes distended to a considerable degree, but still remains motionless and the patient experiences a discomfort described as a great desire to defæcate. The patient is transferred to the Bucky-diaphragm table for the purpose of having a radiogram taken. By this time, that is after the lapse of a few minutes, he complains of severe abdominal pain of a colicky character. The radiogram is taken and the patient returned to the screening table. Strong contractions of large segments of the colon are seen. These contractions synchronize with the patient's spasms of pain which are invariably referred to the suprapubic region, although the contracting segment of bowel, perhaps the proximal path of the transverse or of the descending colon, may be fourteen or fifteen centimetres away. These observations confirm Mackenzie's in demonstrating in the first place the referred component of pain and in the second its production by powerful peristalsis. But their interest at this juncture is the light which they throw upon Hurst's views. Although the bowel is greatly distended, no pain results until powerful contractions of the colon occur. The pain is due, therefore, not to the distension, but to the contractions.

In Bastedo's test for appendicitis[10] the colon is distended with air through a rectal tube; when appendicitis is present, pain and tenderness become evident in the right iliac region. Hurst himself[11] considered this test very reliable. He mentions that in some of his patients adhesions were absent, so that the chief factor is "probably distension of the diseased appendix." Since the distension is produced in the rectum, it cannot be greater in the appendix than in the rest of the colon; therefore, there must be some more important pain-producing factor present in the appendix and not in the rest of the colon. I say more important because this other factor can produce pain in appendicitis without the assistance of Bastedo's distension, whereas the distension alone does not produce pain in normal persons. Apparently the distension causes pain by aggravating this other factor, the nature of which will be discussed later. Bastedo notes that if a large part of the air is not allowed to escape before withdrawal of the tube, colicky pains may come later, just as in the case of the retained enema and they differ in character from the appendical pains. This observation bears out my own in administering opaque clysmas.

During the course of an operation on a conscious patient, Lennander[2] inflated a segment of bowel until it actually burst. No pain was produced.

Consider the changes which take place in acute dilatation of the stomach. The viscus undergoes enormous distension and is so stretched that the "contents are usually visible through the wall." Acute dilatation has occurred during the course of abdominal operations; the stomach is seen to be "drum-like and tense." And yet we find that "the most marked symptom is vomiting." "The pain is usually constant, not severe, being rather a feeling of distension and discomfort than of true pain." The quotations are from Walton.[13]

In post-operative peritonitis the distension may become extreme and cause little or no pain.

The pain and tenderness of a strangulated omental hernia require explanation which is not forthcoming from the theory, that "the only stimulus to visceral pain is distension."

I shall give the stories of two patients who recently came under my notice.

Miss B. complained of obscure digestive symptoms. At laparotomy, the abdomen was seen to be very tense and on being opened, the stomach protruded forcibly like a huge football bladder and occupied the whole of the incision. The bowel also was distended. No organic lesion was discovered. This patient was not in pain when the operation commenced. The distension persisted for about a week after the operation and still the patient did not complain of any pain.

Mrs. K. suffered from pyonephrosis. The affected kidney was removed through a lumbar incision. Next day it was noted that her abdomen had swollen to an enormoue degree and was very tense. No cause could be discovered for the condition which gradually subsided after persisting for some weeks. The patient, in the meantime, did not complain of any pain, but had a smooth convalescence apart from the abdominal and respiratory discomfort associated with her distension.

I shall show later the impossibility of reconciling the distension hypothesis with the peculiarities of ulcer pain.

The foregoing considerations show that it is difficult to produce pain by mere distension of any part of the alimentary canal. If the distension, however, leads to violent peristalsis, pain may be produced and then the pain will follow rather than accompany the distension and it will be colicky in

character. This generalization does not apply to the kidney, pelvis and ureter, for severe pain without any preliminary discomfort can easily be produced when these structures are being filled for pyelography. Apparently the viscera in their reaction to artificial distension vary according to their physiological habits. The kidney, pelvis and ureter are accustomed to a normal pressure of about zero; they do not experience any gross variations in the mass or pressure of their contents and the excursion of their walls in normal peristalsis is small. They are, therefore, not provided with any mechanism or structural adaptability to accommodate sudden distension. Their lumen is small and their walls are relatively thick. The alimentary canal, however, has an altogether dissimilar part to play in the bodily economy. In the course of a normal peristaltic wave its lumen may be alternately obliterated and distended to a circumference of several inches. The stomach may be lying at rest and contracted, but it must be prepared to accommodate itself in a few minutes to the introduction of a meal which will distend it greatly. To meet these conditions the alimentary canal depends on its relatively thin walls and potentially wide lumen, the great adaptability of its muscular tone and the rearrangement which takes place among the muscular fibres, so that the twenty or thirty layers, for example, in the contracted stomach become two or three in the full stomach (Grützner and Müller, quoted by Hurst[12]). Pathological distension of these organs is instructive. In Dietl's crises when the renal pelvis is suddenly distended, dreadful pain is produced. But when the stomach undergoes acute dilatation, pain plays a subordinate part in the symptomatology, as we have already seen. We should expect, therefore, that these organs should react differently to artificial distension, differing as they do in their structure, in their function and in their response to pathological distension. It would be surprising if we had any difficulty in producing pain by distension of the kidney pelvis and ureter and it would be surprising if we could easily produce pain in the alimentary canal by distension, because distension is part of its everyday experience.

Pain may be produced by distension, as in Bastedo's test, when some other factor is already present. We find pain immediately after meals, often during the course of a meal, in patients with carcinoma of the stomach of the widely infiltrating type. But in these cases the essential pain-producing mechanism is not the distension because the distension itself can cause pain only with difficulty and because the pains occur apart altogether from any distension, an aspect which I shall discuss more fully later. I cannot bring myself to look upon distension as the essential cause of pain in those not uncommon cases of appendicitis in which the organ is stiff and thickened and the lumen unobstructed; neither can I consider distension of the whole or part of the stomach as the cause of the pain, when the walls of that organ are infiltrated and stiffened by inflammatory or neoplastic changes.

PEPTIC ULCER.

Facts.

The consideration of pain in peptic ulcer will give the key to the problems. I became interested in the subject of abdominal pain through peptic ulcer, for I was struck by the failure of current theories to explain the pain in this condition.

The pain in peptic ulcer has certain specific attributes which must be explained by any theory of abdominal pain. It is important to define these at the very beginning, because great confusion has arisen, even in the work of eminent investigators through losing sight of these essentials. We are concerned with the pain in persons with ulcer uncomplicated by adhesions, obstruction or malignant degeneration. Although these simple cases include the majority, it is in these that modern theories of pain completely fail. Complications cause alterations in the nature of pain and in the mechanism of its production and will be referred to only in so far as they help us to understand the pain of simple ulcer.

The pains occur one, two or three hours after eating.

Alkalis relieve.

Vomiting relieves.

The pain is subject to intermissions of months' duration, often seasonal, without any healing in the ulcer.

The pain is relieved by food or drink, especially when hot. With regard to the effect of cold water, I specially questioned nine patients with typical ulcer symptoms, in whom ulcer was found at operation. Six reported no pain relief whatsoever by cold water and in three there was slight temporary relief, not nearly so great nor so prolonged as in the case of foods.

I must emphasize the usually steady nature of the pain. Grasp of this premise will save us from error. To establish it firmly, I closely questioned fourteen patients, in whom uncomplicated chronic ulcer had been found at operation. I led them to consider whether there was any undulation in their pain, such as would be expected if stomach movements played a part in its production. Ten agreed that the pain underwent no wave-like fluctuations, but maintained its even tenor, perhaps for hours, until relieved by food or otherwise. One patient described phases in his pain, firstly an ascent, then a plateau, then a descent. The plateau was the most prolonged period and the pain was steady during that time, fluctuations appearing, however, in the ascent and descent. Another patient, a medical man, told me that the pains fluctuated in the daytime, but the night pain was steady. In only one patient was a history of true colicky pain obtained. We can, therefore, accept the proposition that the pain of ulcer is nearly always of a steady character. Occasionally the pain is spasmodic or colicky and in these cases some degree of stenosis is usually present; the pain is more diffuse, is inclined to make the patient double up and is no doubt due to disordered and excessive peristalsis.

When an ulcer heals and stenosis occurs through cicatrization, the pain undergoes important modi-

fications. In the early stages, as has been shown, the pain may be severe and spasmodic or colicky, but later it becomes less definite and "more a constant feeling of discomfort and fullness which is increased by the ingestion of food and comes almost immediately after the food is taken" (Walton[13]). Case I illustrates this modification of the pain.

When the ulcer becomes fixed to neighbouring parts by penetration beyond the stomach wall or by adhesions alone, the pain becomes modified. In advanced fixation (see Case II), it becomes continuous and unrelieved by food or alkalis. In less advanced cases (see Cases III and IV), relief by food and alkalis is only partial. It is remarkable that even a few flimsy adhesions about the ulcer are sometimes sufficient to modify the pain. Penetration or adhesions can thus be predicted from the history alone.

Hurst's Theory.

Brief reference has been made to Mackenzie's and Lennander's views. There remains Hurst's theory of tension or distension.

Hurst considers that pain in peptic ulcer is due to strong peristaltic waves advancing against a pylorus which fails to relax, thus producing a rise in tension in the intermediate segment of stomach; failure of pyloric relaxation and hyperperistalsis are reflexly caused by excess of hydrochloric acid which is particularly effective through acting on nerves "exposed by ulceration"; the onset of pain is some considerable time after food, firstly, because it takes some time for the acidity at the cardiac end of the stomach to extend to the ulcer at the pyloric end where the food is alkaline, secondly, an increase in tone takes place as the bulk of the gastric contents diminishes; food and alkalis relieve because they dilute or neutralize the acid; vomiting relieves because it removes the acid and at the same time empties the stomach, thus leaving nothing upon which the muscular coat can contract. I shall separate the parts of this theory and endeavour to show that each is inconsistent with facts.

The motor phenomena invoked are powerful peristaltic waves advancing against a spastically closed pylorus, the consequent distension in the intermediate segment of gut being the cause of pain. But the experimental difficulty of producing pain by distension of the stomach must be remembered, together with the fact that distension is part of the everyday physiological experience of that organ. It is not uncommon to observe hyperperistalsis and pylorospasm in the absence of pain, even when the patient is afflicted with peptic ulcer. The words of Carman[14] are most significant. "The spastic area is not painful or tender though there may be pain or tenderness localized to the causative lesion." And again: "The spasm (in duodenal ulcer) is rarely or never of the total or regional type. Usually it takes the form of an incisura." It is well known that these *incisuræ* are painless. Pain can occur in peptic ulcer patients whose stomachs manifest normal or diminished movements or even absence of movement, as I myself have observed once. Motor phenomena of all grades, from quiescence to intense activity, and all degrees of tonicity may occur in peptic ulcer patients with or without pain (Reynolds and McClure,[15] Homans,[16] Hardt and Joseph,[17] Wilensky,[18] Carlson,[19] Moynihan,[20] Vandorfy[21]). Even granting for the moment the presence of pylorospasm and strong peristaltic waves, it is difficult to imagine that they could shut off fluid under pressure in a segment of the stomach. The mucous lining is thrown into longitudinal folds which are so soft and slippery that channels through the contracted segment are readily available and may often be seen filled with opaque meal when a radiographic examination is being carried out.

Hyperacid chyme acting on nerve fibres exposed by ulceration plays a part of very doubtful importance. In some cases of duodenal ulcer and in the majority of gastric ulcer not hyperacidity but hypochlorhydria or achlorhydria is found. Moynihan asks: "whether an 'acid' treatment of gastric ulcer would not be more rational than an 'alkaline' treatment." Vandorfy finds hyperchlorhydria in but 50% of a large number of patients. Cases VII and VIII illustrate achlorhydria in patients with ulcer pains. Moreover, it is important to remember that gastric carcinoma is typically associated with hypochlorhydria or achlorhydria and yet ulcer pains may be present (Devine[22]). That acidity should cause failure of pyloric relaxation is an idea born of Cannon's "acid control of the pylorus," a theory now discredited (Devine,[23] Luckhardt, Phillips and Carlson,[24] McBaird, Campbell and Hern,[25] Cole[26]). It is very doubtful whether acid stimulates peristalsis (Hardt,[27] Reynolds and McClure,[15] Carlson,[28] Ginsburg *et alii*[29]).

Even if it be assumed that nerve fibres may be exposed to the acid chyme, it seems improbable that their exposure would play an important part in pain production, in the first place because the floor of the ulcer is composed of two usually non-nervous strata, namely slough and granulation tissue, in the second place because of the analogy of the varicose ulcer in the leg the surface of which I have proved to be insensitive by applying alcohol to such ulcers, without touching the surrounding skin.

Perhaps the most striking evidence against the assumption that acid acts in this manner, was put forward by Hurst himself. He poured 120 cubic centimetres of 0·5% hydrochloric acid into the empty stomach of each of seven patients with peptic ulcer. No pain resulted. Similar observations have been made by Ginsburg *et alii*,[29] Hardt,[27] Reynolds and McClure[15] and myself. Hurst explained this discrepancy between his theory and the facts as follows: "The stimulating effect of the acid on peristalsis and the abnormally prolonged inhibition of pyloric relaxation can only cause pain when food is present in the stomach, as no rise in internal pressure can occur when there is nothing in the stomach upon which the muscular coat can contract." But it is necessary to consider the small, rapidly contracting stomach which is so frequently seen in duodenal ulcer. Pain is at its height four

hours after a meal and by this time the bulk of the food cannot be much more than the 120 cubic centimetres of acid injected by Hurst. In view of the easy distensibility of the stomach, it is difficult to imagine that the presence of a small quantity of fluid chyme, even if assisted by some motor disturbance, could so raise the intragastric tension that severe pain would be produced. Moreover, I have seen a patient who had a hyperacid, hypertonic stomach and a chronic duodenal ulcer, but no pains except for two weeks before a hæmorrhage.

Hurst explains the painless interval after food by the "considerable time that the acid gastric juice (takes to reach) this part to any great extent" and "the increase in tone which occurs as the bulk of the gastric contents diminishes." But we have already seen that pain cannot depend on acid. The "increase in tone" merely means that the muscle fibres have altered their postural length; they have shortened. In spite of Hurst's implication this reaction does not involve any increase in tension, either in the wall or in the lumen of the stomach. On the contrary, it is the function of these tonic variations to prevent alterations in tension.

The relief of pain by food or alkalis is attributed to dilution or neutralization of the acid. Again, we must remember that the acid is harmless, so far as pain is concerned. Moreover, when the ulcer contracts adhesions to neighbouring viscera, food and alkalis do not lose their properties of diluting and neutralizing, but they fail to relieve the pain of the ulcer. I have already referred to the converse condition in which food and alkalis relieve patients with hypoacidity or anacidity. Boas (quoted in reference[29]) reports cases of alkali relief with no increased acid value. In view of these facts, I cannot accept mere physical dilution or chemical reaction as the explanation of food and alkali relief.

Lastly, we must compare the clinical features of ulcer pain with Hurst's theory. Ulcer pains are usually steady, whereas pains depending upon powerful peristalsis are usually paroxysmal or wave-like. The modifications of the pains by adhesions, penetration and hæmorrhage and their seasonal intermissions are very difficult to correlate on the basis of Hurst's theory.

Hurst's theory fails to explain the modification of ulcer pain by adhesions and penetration, their seasonal intermissions and their relief by hæmorrhage.

Ryle's Theory.

According to Ryle's theory of tension and tonus[30] the pain of duodenal ulcer occurs "when the stomach is in posture of approaching emptiness, but through reflexly enhanced tonic action, its muscle becomes and remains abnormally taut. This is relieved when the stomach adapts its posture or relaxes to accommodate introduced food or fluid, or when 'resistance' is withdrawn and intragastric pressure reduced by complete emptying." The pain of gastric ulcer occurs "when the tension in certain muscle fibres, reflexly tautened or structurally shortened by the ulcer, is further augmented by the introduction of food."

I shall enlist the help of Bayliss[31] and briefly consider the fundamental physiology of tone. Tone in smooth muscle "is a general property of this kind of tissue to maintain itself in a certain degree of shortening, apart from impulses from nerve centres." It has been likened to a "catch" or "ratchet" mechanism. The spine of the sea urchin, held in place by a tonic contraction of plain muscle, resists displacement, but once displaced, it tends to retain its new position. The shells of pecten (a bivalve mollusc) may be made to close upon a piece of wood "with so powerful a crash that their edges are splintered. The wood is then held as in a vice. One can, however, pull it out by twisting it about backwards and forwards and then one is surprised to see that the shells remain motionless, just as would the jaws of a vice if an object clamped between them had been forced out." The word "tonus" is inappropriate because "the existence of tension is implied in it, whereas, as we have seen, the varying lengths which a muscle can be made to assume in particular conditions, are quite independent of tension." Tension and tautness are purely mechanical or physical phenomena which refer to the force which tends to stretch a body and to the state of that body when acted on by such a force. The function of tone in hollow organs like the stomach is to enable the organs to assume a posture suitable to its contents, so that tension and tautness do not accompany alterations in volume of the contents.

I shall now consider Ryle's hypothesis, piece by piece. The pain of duodenal ulcer occurs "when the stomach is in a posture of approaching emptiness, but through reflexly enhanced tonic action, its muscle becomes and remains abnormally taut." But tonic action prevents tautness; "enhanced tonic action" should still more effectively prevent tautness. "This (tautness) is relieved when the stomach adapts its posture or relaxes to accommodate introduced food or fluid, or when 'resistance' is withdrawn and intragastric pressure reduced by complete emptying." The "tautness" is, therefore, relieved either by lengthening or shortening the muscle fibre. The phenomena of tonus are ignored altogether, when one speaks of reducing intragastric pressure of emptying. The pain of gastric ulcer occurs "when the tension in certain muscle fibres, reflexly tautened or structurally shortened by the ulcer, is further augmented by the introduction of food." Reflex "tautening" in smooth muscle is unknown to physiologists. The most common manifestation of tonic shortening in the gastric musculature is the *incisura* so well known to radiologists and this is painless. It is of interest to note that extreme tonic disturbances in skeletal muscles, for example in spastic paralysis, are unaccompanied by pain. Ryle makes the presence of pain depend upon alterations in the length of muscle fibres, whether these alterations are caused tonically, mechanically (filling or emptying of the viscus) or pathologically.

Even if these hypothetical alterations in tautness were not contradictory, it would be hard to grasp them in view of the tonic adaptability of the stomach, which easily accommodates itself to considerable alterations in its contents without any increase in pressure. The opposite effects produced by the introduction of food cannot be reconciled: relief of tautness in duodenal and increase in gastric ulcer. I cannot imagine in duodenal ulcer the relief of tautness produced by the introduction and later by the complete evacuation of food. The phases of duodenal pains, a full stomach with no tension and no pain, a partly empty stomach with tension and pain and an empty stomach with no tension and no pain, are difficult to correlate, not only with each other, but also with the gastric pain in which a full stomach causes tension and pain. If the question is merely one of stomach posture or tautness, why is not cold water potent in the relief of ulcer pain? How could a few ounces of milk or a biscuit cause such alterations in the tension of an organ so highly distensible as the stomach? How can the "resistance" of food in the half empty stomach be necessary for pain, seeing that a little more food will relieve the pain? Why do ulcer pains occur in the atonic stomach? Why do they not come immediately after the meal, when tension is most probably greatest on account of the bulk of the meal and the contraction induced in smooth muscle by stretching? What happens to these tonic variations during the weeks of intermissions in the symptoms? How can they explain the symptom modification by stenosis, adhesions, penetration and hæmorrhage?

Even if we could answer these questions and find our way through the maze of alterations in tautness and even if these alterations emerged as facts from among the mass of unproved notions, we should still be faced with the difficulty of transferring the structure of the hypothesis from the muscular to the nervous elements of the stomach wall. Lengthening and shortening are reactions of the muscle fibre and it is difficult to imagine them acting upon the nerve fibres and producing pain. They are atogether different from powerful muscular contractions and tetanic spasms and they are not analogous with the painful cramp in skeletal muscle.

The Kymographic Theory.

Boldyreff in 1905[32] and Cannon and Washburn in 1911[33] noted the contractions which take place in the empty stomach. Carlson[28] [34] [35] confirmed the observations, found that the contractions synchronize with hunger pangs and came to the conclusion that similar contractions caused the pain of peptic ulcer. Ginsburg, Tumpowsky and Hamburger[29] and later Hardt and Joseph[17] supported these views.

Carlson and his followers used the kymographic method of study; their patients swallowed a rubber balloon fixed to the end of an Einhorn tube, the other end of the tube being fixed to a manometer and the usual recording apparatus, which thus produced a tracing of the stomach movements. The conceptions of this school are open to objection for the following reasons.

The conclusions depend very largely upon an analogy with hunger pangs and this analogy is not admissible. Carlson states that ulcer pains were called "hunger-pains" by Moynihan on account of their resemblance to the pangs of hunger, but Moynihan[36] writes as follows, giving a totally different reason: "Many patients will volunteer the statement that the pain begins to appear when they are beginning to feel hungry and I therefore suggested in one of my early papers the term 'hunger-pain' as descriptive of this particular symptom." That the pains in uncomplicated chronic ulcer are quite different from hunger pangs both in their characters and in their mode of production is evident from the following considerations. The hunger pangs and associated stomach contractions are colicky in nature, lasting as a rule for thirty seconds. Occasionally the fusion of several contractions results in tetanic spasm lasting from two to five minutes. Ulcer pains are not colicky and if they depended on this mechanism, it would be necessary to postulate a tetanic spasm lasting for some hours. This spasm must be imagined, for it has never been proved, but on the other hand its absence during pain has often been demonstrated. The tetanic contractions observed and their accompanying hunger pangs were characterized by an abrupt spontaneous cessation after a few minutes, altogether unlike ulcer pains. The hunger contractions and their pains can be inhibited by influences which have no effect on ulcer pains, namely, stimulation of the gustatory nerve endings by sweet, bitter, salt or acid substances, chewing agreeable, disagreeable or indifferent material, smoking, swallowing movements, cold water and two hundred cubic centimetres of 0·5% hydrochloric acid. It is generally agreed that the pains of ulcer occur during the process of digestion and are absent when the stomach is empty. Their persistence or occurrence long after food, for example, in the early hours of the morning, suggest some complications, particularly obstruction. In cases of gastric ulcer the pains disappear before the next meal, hence the "rhythm" described by Moynihan—"food-comfort-pain-relief." Now the hunger pangs were observed too long after food to suggest the pain of uncomplicated ulcer, after four to ten or twenty-four hours by Carlson, after fifteen hours by Rogers and Hardt,[32] and five or six hours after a light meal by Hardt and Joseph.[17]

The cases recorded as ulcer cases do not help us very much, for the pains are not the pains of ulcer. The pains are colicky, like hunger pangs, but unlike ulcer pains. They appear too long after food in most of the graphs. Thus Carlson publishes four graphs showing the "ulcer pains." In three of these the record is made on the empty stomach after a long fast, the patient having had no breakfast when the experiments were performed at about 11 a.m.. Ginsburg and his colleagues do not state the time which elapses between their experiments and the last meal. Many of the cases do not help

ILLUSTRATIONS TO THE ARTICLE BY DR. VICTOR J. KINSELLA.

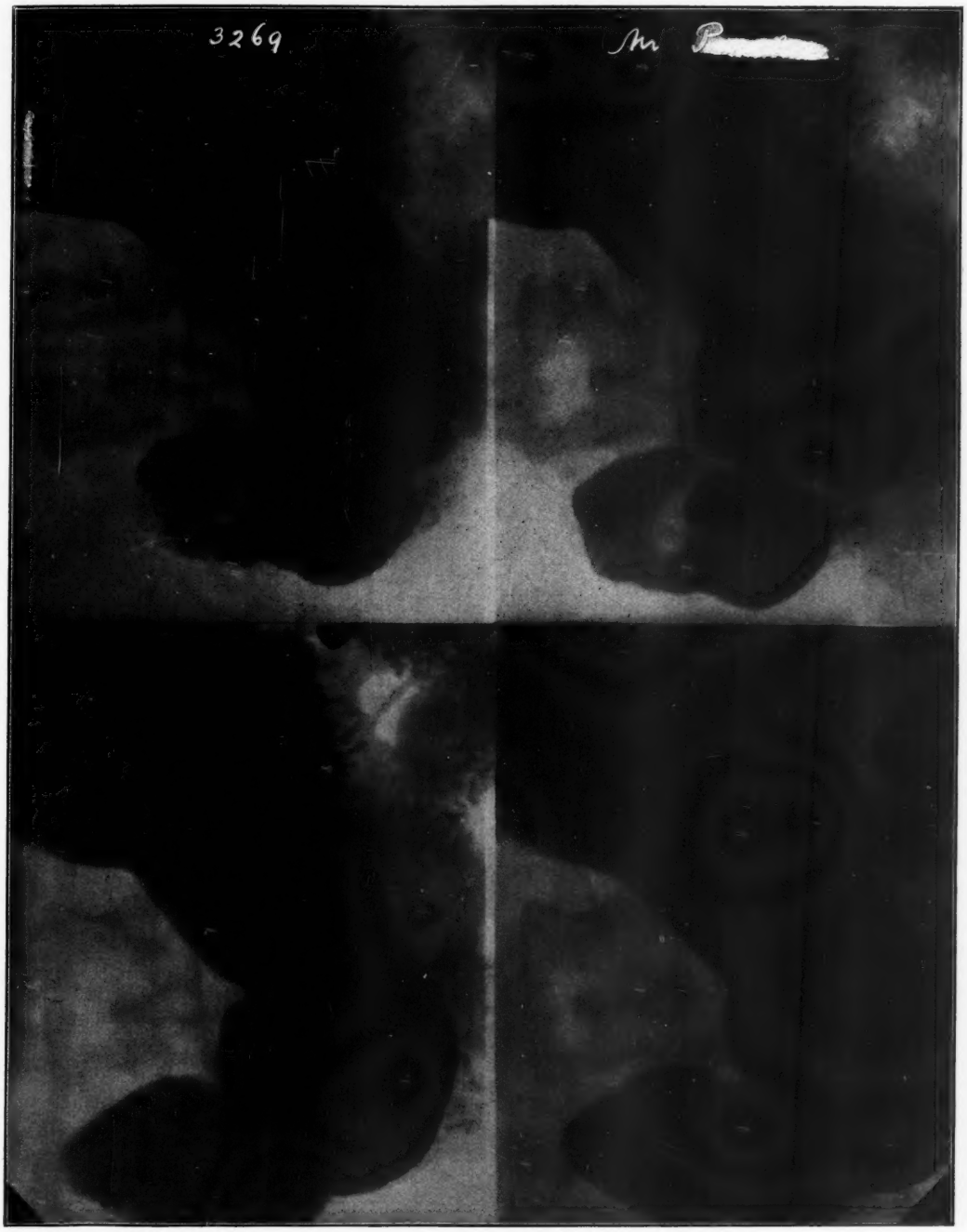


FIGURE III.

Skiagrams showing the relaxing effect of soda on the pylorus and duodenum. The two pictures on the right were taken first, then soda was administered and the two pictures on the left taken after a five minutes' interval.

ILLUSTRATIONS TO THE ARTICLE BY DR. VICTOR J. KINSELLA.

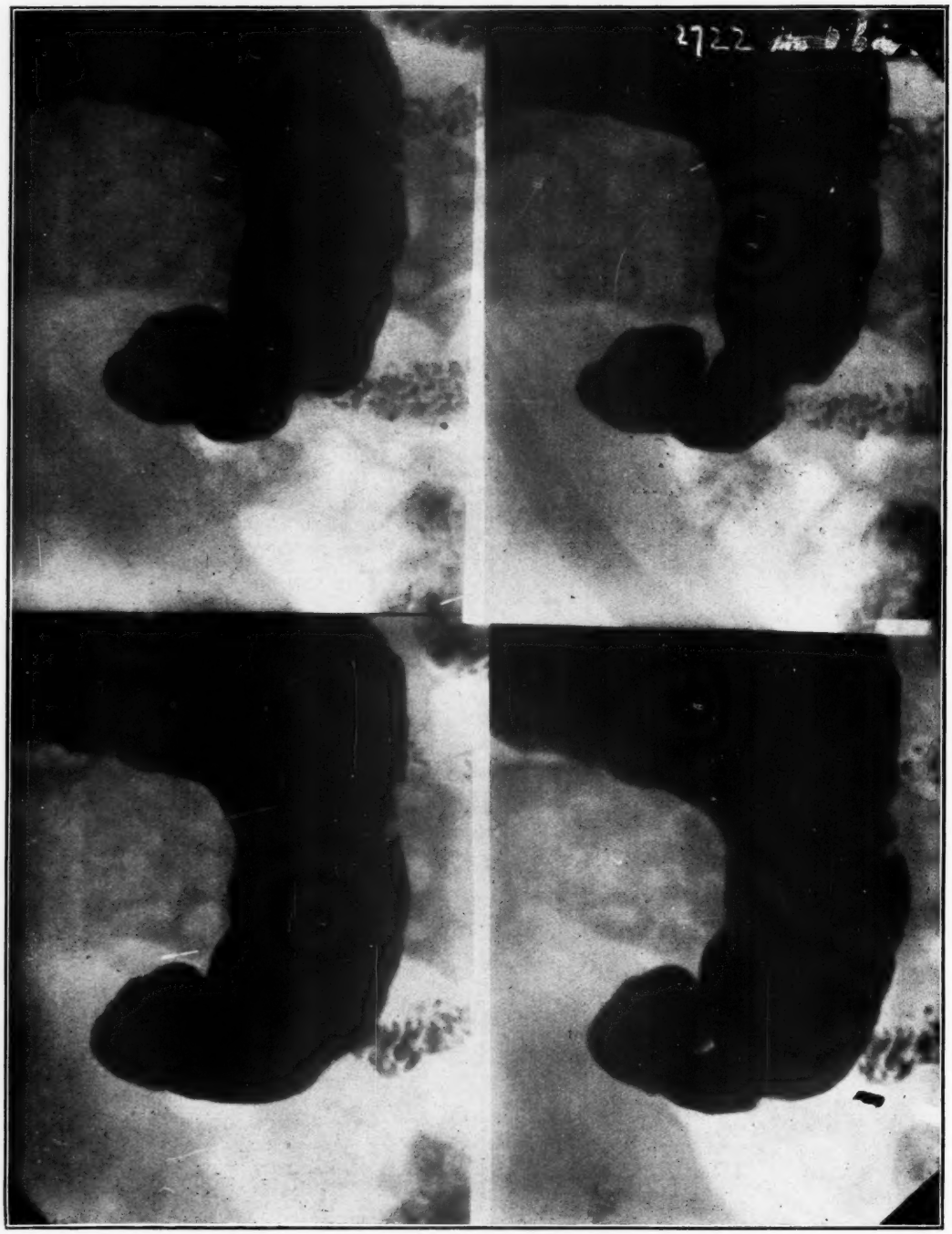


FIGURE IV.

Skiagrams showing the relaxing effect of soda on the pylorus and duodenum. The two pictures on the right were taken first, then soda was administered and the two pictures on the left taken after a five minutes' interval.

us because of complicating factors; the only case quoted in detail by Ginsburg and his collaborators is one of extreme pyloric obstruction with visible peristalsis; of the three patients referred to by Hardt, two had a high grade of pyloric obstruction, in one instance due to inoperable carcinoma. It is quite conceivable that the pains in these cases are due to gastric peristalsis, but the pains are not the pains of ulcer. The diagnosis is open to dispute in each remaining case. In no instance has it been confirmed by operation. In none of the cases investigated by Ginsburg, Tumpowsky and Hamburger and by Hardt is there any mention of radiography, the diagnosis being clinical, except in the stenotic cases mentioned above. In Carlson's solitary patient the radiographic diagnosis was duodenal ulcer, but none of the primary or direct radiographic signs of ulcer were present. In each of the cases detailed there is strong clinical ground for taking exception to the diagnosis. I shall not discuss the cases in detail, but shall content myself with the remark that in each of the patients the pain was colicky in nature.

What are the pains, then, with which these investigators have been dealing? I believe that they are chiefly hunger pangs and partly the pains of advanced pyloric obstruction. But some may depend on ulcer, although it is just as possible that these are part of a dyspepsia either nervous or reflected from some other abdominal lesion. If ulcer does play a part in any particular case, the pains are not typical or even usual.

The contractions said to cause the pains are indistinguishable from the contractions of the healthy stomach. Why then should they produce pain in one case and not in the other? Carlson explains by a "hyperexcitability of the gastric pain nerves in the ulcer patients" and Hardt invokes a mysterious "active hyperæsthetic process," but to my mind these phrases merely beg the question.

In condemning this peristaltic theory I am again encouraged by the fact considered in a previous section, that motor phenomena of all grades, from quiescence to intense activity, have been observed to accompany the pains of peptic ulcer.

Again, this theory fails to explain each of the eight peculiarities of ulcer pains; their steady nature, their delayed onset after meals, their relief by alkalis and so on.

Hardt and Joseph admit the shortcomings of the hypothesis and suggest that "there may be something else besides peristalsis and an adequate acidity necessary to the production of pain, although as yet it has not been definitely demonstrated. We refer to the possibility of a third factor." I shall endeavour to explain the nature of this third factor, but I shall not name it thus, for it is to my mind not the third, but the only essential factor.

The Essential Factor in Visceral Pain is Compression of Nervous Elements.

In visceral pain the depth of the lesion and the number of overlying sensitive layers add to the complexity of the problem; the best plan in seeking for the essential pain-producing factor in disease generally and in peptic ulcer particularly is first to consider the familiar painful conditions in which the parts are more or less accessible and easily studied, for example, whitlow, boils and chronic congestive leg ulcer. In these conditions compression of pain nerves brought about by excessive tension within the tissues is undoubtedly the cause of the painful sensations. Anything which relieves the compression or the tissue tension, whether it be foments, incision or alteration in posture, will relieve the pain. Added pressure, for example by the examining finger, will increase the pain. If we proceed from the somatic to the splanchnic tissues, we find that the principle still holds; thus the painful pus-filled Fallopian tube becomes less painful when tension within the tissues is decreased by hot douches, incision or the flow of menstrual blood and becomes more painful when compressed by the hand of the gynæcologist. Compression of nerve fibres is still the required factor. If we leave inflammatory pain and study colic, we find compression of nerve fibres again the essential factor.

I have endeavoured to avoid the term "tension" used without qualification, because its use in this way has led to confusion on account of the two possible applications of the word. It may mean tension within the lumen of a hollow viscus, which implies a tendency to a forcing apart of the walls and may be more precisely termed distension; this is the "tension" of Hurst which fails, as I have shown, to explain abdominal pain. It may also mean tension within the tissues themselves, involving the wall of the viscus, but not necessarily the lumen and this variety may be aptly termed compression. In a recent lecture[22] Mr. Devine expressed his belief that pain in cancer of the stomach depends upon both these varieties of tension. On the other hand, my interpretation of the evidence suggests that the pain of all organic splanchnic disease depends entirely upon the second variety, namely, compression, and I have already given my reasons for discarding the first variety. Tension causes pain by compressing nerve fibres and tension in the tissues of the bowel wall is the important factor, not tension on the muscular coat, as is usually supposed. This distinction is no mere quibble, but a conception of great importance, because (i) it will enable us to adopt the more secure course of seeking directly for the origin of pain in nervous constituents of the bowel wall rather than seeking indirectly through the muscular elements which, by the way, may be so damaged by painful disease as to be incapable of effective motor activity; (ii) it will bring into line under a common mechanism pain in disease of the solid viscera, pain in disease of the hollow viscera and pain in somatic disease; (iii) it will enable us to account for the peculiarities of peptic ulcer pains in a manner hitherto impossible; (iv)) lastly, and perhaps more important of all, it will enable us to explain "tenderness," that is, the aggravation or production of pain by manipulation. How else can the pressure of a finger tip cause pain in gastric

ulcer than by raising the tissue tension in the inflamed part? Surely not by raising the tension within the lumen of the organ, because pressure outside the localized tender spot, but still over the stomach, has no painful effects; nor by stimulating spasm and powerful peristalsis, for such are not observed by the radiographer to follow painful pressure.

There are two great varieties of compression, which give rise to two great varieties of pain with distinct characteristics: (i) congestive compression and pain, (ii) the compression and pain of muscular contraction.

The congestive variety depends upon vascular congestion and stasis with accompanying increase in volume and rise in pressure of tissue fluids; a cellular infiltration may be superadded. The affected parts accommodate this increase in blood volume, in tissue fluids and in cellular population by swelling until a limit is reached, when compression rises to a degree adequate for pain production and this limit is reached more readily in the more rigid tissues. This congestive type of compression may be due to inflammatory conditions, for example, abscess, or to circulatory deficiencies, for example, the painful liver in some types of heart failure, or to the painful types of malignant disease. The resulting pains are of a continuous, dull character, sometimes throbbing, never paroxysmal or sharp or stabbing or colicky. Perhaps most important of all they are modified by alterations in the circulation, increase in congestion increasing the pain and *vice versa*. Thus the pain of abscess is relieved by a hot application which drains the blood away from the congested into the surrounding tissues. The painful liver in auricular fibrillation soon ceases to cause discomfort when digitalis improves the circulation. The chronic varicose ulcer of the leg becomes painful towards the end of the day when work and standing have played their part, but elevation at once relieves. The dependent and elevated postures influence similarly the pain of whitlow. In the so-called "congestive" or "premenstrual dysmenorrhœa," the vascularity of the premenstrual phase aggravates the congestion in inflamed tissues and the menstrual flow brings relief. Again it is found that pain depending on a definite mechanism has certain definite characteristics.

The second great variety of compression depends upon powerful muscular contraction of hollow viscera. The usual pains of this type are colicky and have already been considered with Mackenzie's work.

It may be asked: "Why is not pain produced by crushing the bowel with forceps, if compression or tension within the tissues be the adequate stimulus?" I shall preface my answer by describing an observation which can easily be made when examining microscopically a drop of urine containing pus or blood. If we try to crush the corpuscles by pressing upon the coverslip, they rush about across the field, but their structure is unaffected. We know that they are protected by the surface tension of the fluid and we may press until the coverslip breaks and still fail to crush the delicate cells. Similarly the bowel is in structure so labile that it is impossible by gross mechanical action to bring about a compression of the cellular elements or of the nerve fibres as they lie in their slippery fluid matrix. This can easily be shown by thoroughly crushing the middle of a normal freshly removed appendix by repeated pressure with artery forceps. The mucous and muscular coats are broken and their ends forced apart within the intact serous sleeve. They have not been compressed, but fragmented, as though they were apple jelly and when the forceps are removed, the broken ends can be made slide back into place and meet readily, for their substance has not been destroyed. The "crushing" therefore, has not compressed the nervous tissues, but has divided them as though by tearing, thus causing a disturbance of the variety which does not biologically concern the viscera. Could the crushing be carried out on the bowel thickened and rigid through morbid changes, the results doubtless would be different, along the lines indicated by the tenderness of inflamed organs on clinical examination, and by Finsterer's experience of laparotomy with local anæsthesia. Thus when Mr. Devine crushed the base of an appendix whose texture was altered by inflammation, the patient complained of pain and vomited.[22]

In order to demonstrate pain production by cellular compression I experimented upon the loop of colon exposed for colostomy in five patients with carcinoma of the rectum. The time chosen was twenty-four hours after the operation, in order to allow the patient to recoved from the anæsthetic, further delay being inadvisable on account of the regressive changes which take place in the exposed tissues. The bowel was pinched with forceps strongly enough to damage it to a slight extent. No pain resulted. A needle was then inserted into the muscular coat and warm normal saline solution was injected suddenly and forcibly. In the first four patients a hypodermic needle and syringe were used. In three of them no pain resulted, but in one slight pain referred to the hypogastrium accompanied each injection. In these patients the fluid was injected too gently and produced little or no hardness in the tissue, so I resolved to use a wide bore needle and a ten cubic centimetre syringe, but up to the present have only had one opportunity of so doing. This fifth patient was given ethylene-oxygen anæsthesia and was therefore very suitable for the experiment, which could be carried out within twenty-four hours of the operation. The bowel was strongly pinched by forceps without causing pain. A syringeful of normal saline solution, that is about twelve cubic centimetres, was rapidly forced into the bowel wall which became stiff like an acutely inflamed, œdematous appendix. Immediate pain resulted. This was referred to the hypogastrium and was at first associated with a feeling like the call to defæcation. A gentle pinch with the forceps produced no effect. A firm pinch

in the injected area caused aggravation of the pain which became severe, but was relieved by removal of the pressure. A similar pinch caused no pain when applied to an uninjected part of the bowel. This pain was severe and steady for about five minutes and then gradually subsided as the saline solution became absorbed and the bowel softened. It must have been due to the compression of splanchnic nerves by the infiltration, because care was taken to avoid disturbing the skin incision and no peristalsis was seen to pass over the loop.

The Author's Congestive Theory.

The steady nature of the pain in peptic ulcer suggests a steady cause and therefore I came to the conclusions that congestion, rather than disordered motor phenomena, is the adequate stimulus for which we have been seeking. On further consideration this conception was found to explain the facts satisfactorily. As will have been seen, the essentials for compression of the congestive type are first, hyperæmia and secondly, tissue which is unyielding or has yielded to its limit.

The hyperæmia in peptic ulcer, as in many other inflammatory conditions, for example, chronic salpingitis and varicose ulcer, is of twofold nature, a pathological congestion continually present, being aggravated during certain periods by functional hyperæmia. The two together are more than can be painlessly accommodated by the unyielding tissue.

Many observers have demonstrated by plethysmographic and other studies the enormous increase in the circulation of glandular and muscular organs which takes place when these are functionally active. Thus Kaufmann[37] measured "the blood flow through the masseter muscle and the elevator of the lip in a horse in which the muscles were exercised normally and by the act of eating. The blood flow was increased as much as five times over that during rest and that this is due in part at least to a local dilatation, seems to be proved by the fact that the blood pressure in the artery fell, while that in the vein rose." With regard to the stomach, Beaumont noted that "on the application of aliment the action of the vessels increased, the colour brightened." The part played by physiological hyperæmia in the pain of peptic ulcer is suggested in many ways. I have noted the fact that pains occur during digestion and are absent when the stomach is normally empty, so that obstruction is suggested if the pain occurs late. Then, if the ulcerated stomach be filled with some material which fails to excite digestion, although it excites movement, pain will not result. Time and again during radiographic examination I have detained patients with peptic ulcer in the hope of watching their stomachs during pain. Only twice have I had the opportunity, although I waited long past the free interval after food which these patients usually enjoyed. The explanation is to be found in the nature of their meal, water, barium sulphate and tragacanth, which brought into play the motor functions of their stomach, but did not cause pain because it failed to evoke the physiological hyperæmia of digestion. Reynolds and McLure, who observed patients in pain after an opaque meal, administered one hundred and sixty grammes of finely ground, lean beef and forty grammes of barium sulphate baked in a loaf. Furthermore, the ulcer pains are not relieved by the cold water, but they are best relieved by warm food either solid or liquid, the effects being proportional to the effects on the gastric circulation.

The part played by the second factor, namely, unyielding rigid tissue, is proved by the acute ulcer which of itself causes no pain, and "gives no other indication of its presence than hæmorrhage or perforation" (Moynihan[38]). The motor phenomena, the acid chyme and the exposed nerve endings are there, but no pain. There is no infiltration, no rigidity in the tissues and no possibility of congestion producing compression of nerve fibres.

The essential points are, therefore, congestion in indurated tissue, aggravated by functional hyperæmia. They were illustrated in a patient whom I saw recently, with a myoma as large as an average orange, projecting from the stomach wall on its serous aspect. An ulcer had developed at the attached base of this tumour. The symptoms were pains indistinguishable from those of ulcer. When the tumour was removed the pains disappeared, but examination showed the stomach movements and tonicity to be unaltered. We must, therefore, look to the diseased area for the origin of the pain, not to the motor phenomena. I shall now endeavour to account for the clinical peculiarities of ulcer pain.

The delayed onset of the pains after food and their relief by food are explained as follows. Increase in blood supply occurs in the stomach after a meal, the whole capillary bed is opened wide and the ulcerated area is not unduly congested. As the stomach empties, contraction of the capillary area takes place, the stream bed becomes diminished, but not uniformly. The indurated ulcer cannot partake in the general retraction of the stomach wall and more blood flows into its thickened, rigid vessels as neighbouring channels become narrowed. The healthy stomach wall plays the part of a contractile sponge which squeezes its fluid contents into the non-contractile ulcer. In Figure I, I have represented my idea diagrammatically. A represents one one of the main arteries, for example, the right gastric, and B is one of its branches; C is the capillaries in the indurated area; D is the capillaries in neighbouring normal stomach wall. With the aid of some glass and rubber tubing and a douche can to produce a pressure head, the hydraulic principle involved can be illustrated by a simple experiment. The calibre of A and B remains practically constant and the flow is varied only by alterations in the terminal branches; C and D are variable, their calibre becoming decreased as the stomach

empties. The capillary bed is compressed by the retraction of the stomach as it empties, just as in the case of the uterus. The flow through the vessels is proportionately decreased. But when the area represented by C becomes rigid and non-contractile, the capillary area D alone decreases as the stomach empties, increased blood flow results in C and can be illustrated experimentally by replacing tube D by a smaller tube D^1 and measuring the flow through C. The flow in C is increased the more when the

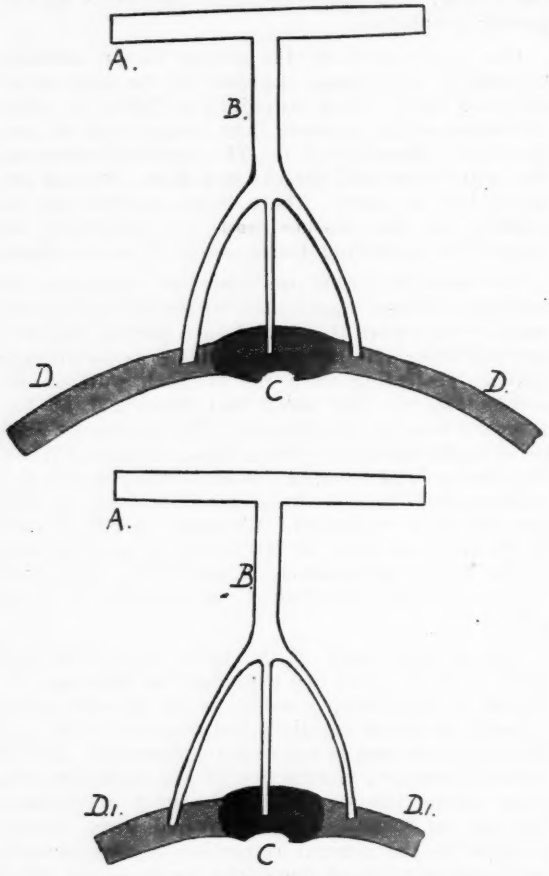


FIGURE I.

Diagram to illustrate the increased congestion brought about in the rigid ulcer by retraction of the stomach. The upper drawing represents the condition in the full stomach; the lower, in the stomach in process of emptying. Degrees of vascularity are represented by depth of shading. A, one of the main gastric arteries; B, one of its branches going to the stomach; C, the capillary bed of the diseased tissue; DD, the capillary bed of the full stomach, becomes D_1D_1, the capillary bed of the emptying stomach.

original difference between C and D is great and when the difference between D and D^1 is great. Thus, in the case of the stomach, the area of healthy compressible capillary bed is relatively great compared with the ulcer capillary bed and the decrease in the healthy capillary area is great as the stomach passes from its full to its empty phase; the physiological and pathological conditions aid the hydraulic factor. But, if the infiltration spreads, as in carcinoma, C becomes greater and D becomes less and the contractility of D (that is, the difference between D and D^1) becomes less and the circulatory fluctuations in C become less. Clinically it would be expected, as in fact occurs, that food relief becomes less definite, until eventually pain appears immediately on eating. The relief obtained by taking food, especially hot food, can therefore be understood. The stomach expands, the digestive processes with their associated hyperæmia are stimulated (that is, the flow in D becomes greater) and blood is drained away from the congested ulcer C into surrounding healthy tissue. The mechanism of relief is parallel to the application of a foment to a boil and similar to the menstrual flow which relieves the pain of dysmenorrhœa.

The Action of Alkalis.

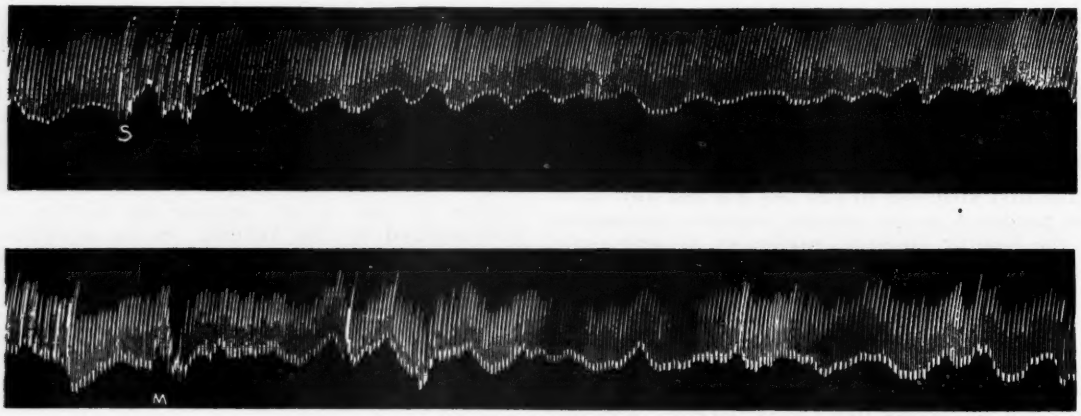
It is well known that alkalis relieve the pain of peptic ulcer. Poulton[39] states that this property is due to liberation of carbon dioxide which stimulates contraction and so prevents stretching of the muscle fibres. But I cannot accept this view, because magnesia relieves as well as carbonates, but liberates no carbon dioxide. The carbonates may be more popular than magnesia, because the belching of wind affords great satisfaction to many patients, particularly when a nervous element is present. It is further well known that hydrochloric acid introduced into the ulcerated stomach causes no pain. Hurst showed this in seven patients and the observation has been confirmed by others. In one patient in whom the diagnosis of gastric ulcer was confirmed at operation, I poured one hundred and twenty cubic centimetres of 0·5% hydrochloric acid into the stomach and no pain resulted. The relief of pain by alkalis and the failure of acid to cause pain are facts irreconcilable by current theories according to which pain is held to be due to hyperperistalsis and spasm and alkali relief is attributed to neutralization of acid. I have already considered Hurst's attempt to adjust this contradiction. Ginsburg and his collaborators attack the problem from a similar point of view.

Such confusion is unavoidable if neutralization of acid, a mere chemical property of alkalis, is relied on to explain the relief of ulcer pains. When an ulcer has penetrated a viscus which is itself insensitive, alkalis fail to give relief. The inflammatory extension cannot interfere with the chemical attributes of these compounds; some other property must be sought to explain the effects. I consulted Cushny's textbook of pharmacology, but learned that the "effects (of the alkalis) on the movements of the stomach require further investigation." Ginsburg and his colleagues in 1916 stated that "1% solution of soda in 300 cubic centimetres quantity, produced no noticeable effect" on the stomach movement, but the details and the extent of their experiment are not given. Hardt in 1918 published a case in which he had relieved pain by alkali; the stomach became comparatively quiescent, although in the published kymograph there appears little difference before and after administration of

the alkali. Reynolds and McLure in 1922 described the condition of four patients in whom they relieved pain by alkalis; in three of these "abnormal motor phenomena . . . . disappeared," but in the fourth no effect was produced. The inconclusive nature of this evidence suggested a further study of the alkalis.

The first step was to find out whether any effect was produced on the normal digestive movements of the healthy stomach, preferably of orthotonic character with contents of average acidity, in a subject who had never been afflicted by dyspeptic symptoms. These requirements are fulfilled in myself, so I commenced a kymographic study of my own stomach. A delicate rubber balloon about twelve and a half centimetres in length was attached to the end of an Einhorn tube and swallowed. The tube was attached by the other end to a Marey tambour and through a T-piece air was injected until the membrane of the tambour became slightly convex. Graphic records were then made on a revolving drum in the usual manner. The balloon was watched fluoroscopically and an attempt made to place it in the pylorus, but, as previous workers had found, it preferred to remain in the most dependent part of the stomach, not quite in the pylorus. It must be clearly understood that this apparatus merely records intraabdominal tension and the waves are superimposed upon this base line. Anything which influences intraabdominal tension, produces its effect upon the graph. Its value as far as the stomach goes is therefore not in its absolute, but in its relative variations. In other words, while it can record peristalsis, it cannot record alterations in gastric tone. If a postural lengthening or relaxation of the gastric musculature occurred, this could not be expected to decrease intraabdominal pressure and lower the graph towards its base line. Hence, the fallacy of a comparison between graphs from the point of view of gastric tension and tonus, such as Hardt and Joseph have attempted. The work was carried out between two and five hours after meals when gastric digestion was at its height. Ten or fifteen minutes were allowed to elapse before observations were made, in order that the stomach could get over any influence which the swallowing of the balloon may have had. The movements were recorded for ten to fifteen minutes, then the substance to be tested was swallowed and the movements recorded for a further fifteen to thirty minutes. This period was considered sufficient for my purposes, because alkalis relieve the pain of ulcer in four or five minutes. Graphs are shown in Figure II. Attention is first attracted by the conspicuous church-steeple waves which are due to respiration and appear about twelve times per minute. In between each of these at their bases a little irregularity is noted which on close inspection is seen to consist of small waves, made plain by accelerating the drum. These represent aortic pulsations and are of value in proving the sensitiveness of the apparatus. The gastric movements are represented by long shallow undulations that are best read along the lower edge of the tracing and occur at the rate of one or two per minute. Sudden irregular excursions of the graph are caused by anything which influences the pressure within the abdomen, for example, movements of the body, coughs, sighs, pressure of the hand on the abdomen and so on. Nine experiments were made with fifteen to thirty cubic centimetres of cold water, because the alkalis were administered in this vehicle. Seven experiments were made with a small teaspoonful of sodium bicarbonate and eight with two and a half grammes of magnesia. Sometimes a brief rise in the tracing followed the ingestion of soda, due no doubt to the evolution of carbon dioxide and this subsided as the gastric muscle accommodated its tone to its increased contents. Occasionally brief spontaneous increase or decrease in gastric activity occurred, sometimes before, sometimes after administration of the water or powder. But neither the water nor the alkalis had any definite effect on gastric peristalsis.

My next line of inquiry was in the routine X ray examination of those patients who presented themselves with dyspeptic symptoms. Of course, these are a miscellaneous group, numbering about a hundred and including patients with functional and organic diseases and some with no disease at all. They were given a meal of barium sulphate suspended in water with mucilage of tragacanth and the routine observations were made. Then a small teaspoonful of sodium bicarbonate in about fifteen cubic centimetres of water was administered and the observations continued. With the subject in the prone position skiagrams were sometimes made, four on each film, two before administration of the soda and two four or five minutes after. The target-film distance and relative positions were constant. Movement of the subject was avoided by giving the soda through a funnel and tubing. Two facts were determined: (i) sodium bicarbonate in therapeutic doses does not diminish gastric peristalsis; (ii) it relaxes the pylorus and the first part of the duodenum. Peristalsis interferes with the radiographic demonstration of the relaxing effect which can be demonstrated better by a fluoroscopic examination. The pylorus and duodenum appear fuller and broader and when palpated they give an impression of relaxation, meal being more readily forced into the pylorus and through the sphincter. In 75% to 80% of the composite skiagrams the relaxing effect is masked by peristalsis, but it is obvious in the remainder in which four exposures were made in a comparatively quiescent stage. Figure III illustrates this. The increased fullness and broadness of the pylorus and duodenum are evident in spite of the fact that less meal was present in the stomach when the second two skiagrams were taken. Sometimes it is difficult to fill the duodenum, but after the soda this difficulty may disappear entirely and the bulb fills readily and spontaneously. The meal may be seen pressed against the pylorus by a moderate wave, but very



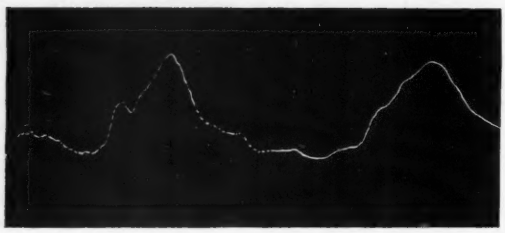


FIGURE II.

Typical kymographic tracings.

A. Before and after soda, administered at S.
B. Before and after magnesia, administered at M.
C. A tracing taken when the drum was accelerated to show more clearly the small waves due to aortic pulsations.

little passes through and the duodenum is imperfectly filled; but after the soda, similar peristalsis has no difficulty in pressing the meal through a relaxed pylorus and the duodenal bulb is well filled. The relaxation of the pyloric sphincter by sodium bicarbonate was noted by Carman and this substance is recommended to assist in filling the duodenum. My observations suggest that not only is the sphincter relaxed, but that the whole of the pyloric and duodenal musculature is also relaxed. This relaxation explains the more rapid emptying of the stomach brought about by soda, which was demonstrated in gastric analyses by Spencer, Meyer, Rehfuss and Hawk,[40] Apperly.[49] [50]

An explanation of the alkali relief of ulcer pains is now more accessible. It is known that depression of peristalsis plays no part. I believe that there are two factors at work. In the first place there is the relaxation of the pyloro-duodenal musculature, which will relieve compression and pain by loosening the muscular grasp upon the ulcer and by opening up the capillary bed in the healthy stomach wall and so draining blood from the ulcer as is done by food. I have produced direct evidence to support this view. The second factor is a mild hyperæmia of the stomach wall possibly brought about by the alkalis and acting as a foment on the ulcer. Direct evidence of this is lacking, but Spencer, Meyer and Rehfuss and Hawk[40] have shown that soda stimulates the gastric secretion and it is known that increased glandular activity is associated with increased blood supply. Relief by alkalis is clinically so similar to relief by food that I would expect to find on the application of soda, as Beaumont found on the application of aliment, that the action of the vessels is increased and the colour brightened.

Other Features of Ulcer Pains.

The relief by vomiting is inexplicable by current theories of motor disturbance. Hurst states that it is on account of the removal of acid and of material upon which the muscular coat can contract. But it has been shown that acid is not the cause of the pain and that the bulk of contents, for example, in barium meals, has no influence in producing pain. Carlson finds that strong gastric contractions, like hunger contractions, accompany vomiting and he is faced with the difficulty of explaining why these contractions should sometimes cause hunger pangs and yet produce no sensation when associated with vomiting. He says: "It is possible that the change in the physiological conditions of the gastric nervous mechanism usually present in the vomiting may account for the absence of hunger." Similarly, the relief of ulcer pain might be explained in this way, but I think that my conception will leave us upon more solid ground. Vomiting empties the stomach and so brings to an end gastric digestion. The physiological hyperæmia disappears and with it the adequate congestion and therefore the pain. Similarly, the pain of premenstrual dysmenorrhœa disappears when the physiological hyperæmia of the premenstrual phase is relieved and in like manner the varicose ulcer, painful at the end of the day, ceases to give trouble when the hyperæmia of the hard-worked leg is diminished by elevation of the part.

If congestion be the cause of ulcer pains, then cicatrization and healing with their decrease in pathological vascularity should bring about an amelioration of the pain. This is found to be the case. It is well known to surgeons that ulcer scars are unaccompanied by pain. When the healing produces pyloric stenosis, the ulcer

pains become less prominent in the symptom picture, in spite of exaggerated peristalsis and their place is taken by a different sensation, not so much a pain, but a constant feeling of fullness and discomfort which is immediately aggravated by food. Case I in the appendix illustrates these facts. If on the other hand pylorospasms and powerful peristalsis were the usual cause of ulcer pains, when stenosis begins, an increase in the severity of the pain but no alteration in its character would be expected. In some unusual cases severe pain has occurred, depending on powerful peristalsis, but the pains have been colicky, unlike the pains either of ulcer or of stenosis (Walton[13]).

When the ulcer contracts adhesions to the bowel, abdominal walls, omentum, liver or pancreas and penetrates beyond the stomach wall, a modification in the symptoms takes place. In those extreme cases when the pancreas or liver form the floor of the ulcer, the pain becomes continuous and there is no relief by food or alkalis. Before this stage, when adhesions have formed, food and alkali still relieve but only partially and the period of relief becomes shorter. The patient thus tells us that while he was in the habit of relieving his pain for many years by a small dose of some favourite medicine, this began to lose its effect, so that finally he gave up taking it. Three illustrative cases (II, III and IV) have been detailed in the appendix and Friedman has recorded others.[41] The explanation is that the ulcer has now obtained a secondary vascular connexion which is independent of the gastric circulation and so uninfluenced by food or soda. Moreover, the adhesions interfere mechanically with the relaxation which should take place when food or alkalis are administered. When the parietal peritoneum is involved, another factor, the sensibility of this layer, is, of course, introduced.

I think that Moynihan was the first to draw attention to the seasonal incidence of ulcer pains. In the symptom-free interval the ulcer is not healed, as has often been proved by operation. Whence comes the relief of symptoms? It would be hard to solve this problem by appeal to gastric motility because motor variations do not correspond with the presence or absence of pain. Therefore these intermissions are a powerful argument against the theories propounded by Hurst and Ryle and the kymographic school. The movements go on as before, the ulcer and hydrochloric acid are still present, but the pain has gone. Probably the ulcer attack corresponds with an increase in the inflammatory changes in the indurated ulcer area and so the vascular congestion and the compression of nerve fibres reach a degree adequate for pain production. Rosenow[42] points out that these exacerbations occur when infection of the nose and throat is prevalent and probably depend upon reinfection from the septic focus by the same strain of organism with its elective affinity for the stomach and duodenum. Such exacerbations are well known in chronic inflammatory conditions other than ulcer, for example, in bronchitis, tonsilitis, pyelitis, salpingitis and in many others. Sometimes aggravation of the ulcer symptoms occurs before perforation takes place and the conclusion cannot be resisted that both the alteration in the symptoms and in the subsequent perforation depend upon an acute exacerbation of the chronic inflammatory changes. In the Museum of Melbourne Hospital there is a specimen (number 7·39) illustrating this type of lesion.

I shall describe in some detail the case of Mr. O'D., because it first suggested a point of view which helps to solve our problems.

The patient was an intelligent man, thirty-four years of age, who had had typical peptic ulcer symptoms from the unusually early age of nine. His epigastric pain was completely relieved by food or hot drinks for two or three hours; it was subject to exacerbations and could be relieved by lying down, but showed no wave-like character. Cold water gave occasional slight relief for a few minutes, but as a rule it failed entirely. Seven years before I saw the patient he had had a duodenal ulcer excised, but his previous symptoms returned twelve months after the operation. In recent years he suffered from six large hæmorrhages, the last of which occurred two months before I saw him. He was able to predict the hæmorrhages, because for several days before each the pain became continuous and much more severe, so that nothing relieved it, food and the usual alkali losing their customary effect. But after the hæmorrhage the pain always disappeared. On the last occasion the patient was in great pain at nine o'clock in the morning; this gradually passed off, its place being taken by nausea, so that at eleven o'clock the pain had disappeared, but the nausea was worse. Early next morning he vomited large quantities of dark blood and the stools became black. Operation revealed a recurrent ulcer of the anterior wall of the duodenum, not involving any macroscopic artery. Apparently excessive congestion defying the usual mechanism of relief had yielded to a gradual oozing from the floor of the ulcer, even as congestive dysmenorrhœa is relieved by the menstrual flow. Nausea was caused by gradual distension of the stomach with extravasated blood. The bleeding was not sufficiently profuse to cause severe general symptoms of sudden blood loss.

The relief of pain in this patient cannot be attributed to the diluting and alkalizing effect of the blood in the stomach, because no relief followed the ingestion of food and alkalis. The only logical outlook is to consider the pain in the light of congestion. Six patients in whom chronic ulcer had been found at operation, were especially questioned with regard to their symptoms before and after hæmorrhage. Four reported aggravation of pain before and relief after; in a fifth there was no pain at any stage of the disease except for two weeks before the hæmorrhage; and in the sixth the hæmorrhage did not influence the symptoms. When hæmorrhage occurs as a gradual oozing from the surface of the ulcer, a modification of the symptoms may be expected, but when it comes from erosion of a large vessel, the blood supply not of ulcer but of some distant part of the stomach wall is tapped and severe blood loss dominates the clinical picture.

Of interest is the symptom-rhythm described by Moynihan[20]: quadruple in gastric ulcer, "food-comfort-pain-relief"; triple in duodenal, "food-comfort-pain," the pain disappearing before the next

meal in the one, but not in the other. As a general rule, the nearer the ulcer to the cardia, the shorter is the free interval after food and the shorter the duration of the pain. When the ulcer is nearer the duodenum these periods become longer. Hurst's theory associates the quicker onset of pains in lesions of the cardiac end with the secretion of acid by this region, but it offers no explanation of their quicker disappearance. The acid is more concentrated at the cardiac end, not only in the early stages of digestion, but even after two or three hours. I have been able to demonstrate this fact in several patients and hope to give a more detailed account in a subsequent communication. But the difficulty is overcome by realizing that ulcer pains begin when the stomach in the process of emptying contracts upon the hyperæmic capillary bed in the neighbourhood of the ulcer, thus augmenting the congestion in the fibrosed area, until the compression produces pain. When digestion ends, the physiological hyperæmia disappears and relief follows. The cycle commences earlier and ends earlier in the cardiac end of the stomach, because this part empties first, at least in the hypotonic type of stomach generally associated with gastric ulcer.

Three patients in my series of fourteen with non-obstructive ulcers described a wave-like quality in their pains. One of these described three phases in his pain, first the ascent, then the plateau, then the descent. The plateau was the most prolonged period and the pain was steady during that time, the wave-like character appearing only in the ascent and descent. Another of these patients was a medical man and his pain was colicky in the daytime and steady at night. Radiographic evidence is lacking in two of these patients, but in the third it revealed the hyperperistaltic type of stomach, which was probably present also in the other two patients because they are of the hyperæsthenic habitus and had duodenal ulcer. Just as a light squeeze with my forceps failed to aggravate the pain in the patient with the exposed loop of colon, so the usual peristaltic wave fails to effect the pain of ulcer, but stronger waves, like a stronger squeeze, aggravate the compression and cause pain.

Occasionally the pain in appendicitis and gall bladder disease comes on at intervals after food and simulates ulcer pain. But the onset is always more erratic and the free interval after food less constant, sometimes lasting a few minutes, at other times an hour or two. I have questioned three patients whose hunger pains were proved to depend upon an inflamed appendix. The proof lay in the failure to find at laparatomy any other cause of the pain and in the permanent cure of the pain by removal of the appendix. Two out of the three agreed that the pain was steady. Hurst and his school have explained these cases by postulating a reflex hyperchlorhydria with gastric spasm and hypermotility. But the difficulties which confronted this explanation in true ulcer, still remain in the case of simulated ulcer. These reflex gastric disturbances are not found in the majority of cases (see Case VI) and very often, when found, they are unaccompanied by pain. The pyloric spasms observed by Moynihan in these cases is transitory, lasting but a few minutes and it is hard to accept with Moynihan[45] a "tumult of peristaltic activity" as a cause of steady sustained pain. Seeing that the ulcer and pseudo-ulcer pains are so similar, their origin must be sought at a common source. In these reflex cases, is there any inflamed tissue in the neighbourhood of the stomach resembling the indurated ulcer tissue, unable to relax during digestion and with its blood supply within the sphere of influence of the vascular changes in digestion? Moynihan[38] has described certain "gastric stigmata" which enable the surgeon to recognize inflammation of the appendix before seeing that organ. These are spasm, "the pyloric blush" and the enlargement of glands on the greater curvature. The two latter depend upon a lymphangitis arising from the inflamed appendix. The path of infection has been studied by Braithwaite,[43] Bercenau[44] and others. Similarly in association with gall bladder disease, a lymphangitis and a lymphadinitis are found in the stomach neighbourhood, mainly along the lesser curvature. The importance of this has been emphasized by Deaver and Pfeiffer.[45] These observations offer a possible pathological basis for pseudo-ulcer pains. In 1925 Ian Schoemaker definitely correlated these pains with the operative findings, when he described forty-five cases of what he calls "the red stomach," in which there was intense hyperæmia of the serosa and swollen hyperæmic glands along the greater curvature, in patients whose symptoms were indistinguishable from those of ulcer.[46] Unfortunately, no note was made about the glands at the time of operation in my patients, but in all three definite tenderness in the pyloro-duodenal region had been noted at fluoroscopic examination. I believe that the epigastric pain and tenderness in these patients are analagous to the inguinal pain and tenderness when the inguinal glands are infected through inflammatory conditions of the leg.

There now remains for discussion a few miscellaneous features of pain which must be examined in the light of my compression theory. Malignant growths are instructive. When the tension within the tissues is raised by their invasion and by the inflammatory reaction, pain results. It is severe in the hard, rapidly-growing tumours which infiltrate rather than destroy. In other types of growth, for example, rodent ulcer, destruction of tissue without infiltration is the outstanding feature and no pain results. Again, there is no pain where surface proliferation and fungation take place. The determining factor in pain production is compression of nerve fibres. The fungating carcinoma of the stomach is "silent" and the hard infiltrating type is painful.[22] Pain is inclined to follow food immediately on account of the digestive hyperæmia. Relief by food is less complete and less prolonged or

altogether absent because the extent of tissue rigidity mechanically prevents relaxation and deflexion of the blood flow from the diseased area. It might be expected in its early stages before extensive infiltration occurs that the malignant ulcer would resemble the simple ulcer in symptomatology; this is sometimes the case and the microscope may be necessary to distinguish them.

Strangulated omental hernia emphasizes the weakness of hypotheses which depend upon muscular reactions. The pain and tenderness in the herniated mass can be better explained by congestion and the consequent compression of nerve fibres.

In cases of severe renal or biliary colic the pain may be of a dual nature, well described from personal experience by Osler. As well as the wave-like or intermittent pain of colicky nature, there is sometimes a constant dull pain. The first is due to compression of nerve fibres by powerful peristalsis and it often radiates. The second type, the dull pain, is localized and the compression is due to an inflammatory reaction set up by the stone and to a prolonged spasm of the muscle fibres in immediate relation to it. The importance of this spasm in smooth muscle endeavouring to expel a foreign body has been demonstrated in intestinal obstruction by gall stones, in which case the spasm is the main obstructive factor (Barnard[47]). In these circumstances the two varieties of compression, the congestive and the muscular, are associated, sometimes one, sometimes the other predominating.

Conclusion.

The subject matter of my paper is not only of academic interest, but of practical importance, for our outlook upon pain production will influence our clinical investigations. I remember a patient complaining of pain and tenderness deep beneath the costal margin which proved later to depend on renal calculus. At that time my outlook was limited by my reading of Mackenzie's conceptions and I failed to make the correct diagnosis because the pain was not within the typical area of referred renal symptoms; it was, in fact, truly visceral pain. On another occasion I saw a patient with right iliac pain and made an uneasy diagnosis of appendicitis, feeling at the time that the pains were unlike the pains in this condition. My habit of thinking in terms of Mackenzie's theories prevented me from considering the gall bladder as the explanation of pains so low in the abdomen. Operation revealed a normal appendix and a very low gall bladder, full of stones.

I cannot do better than quote in conclusion the following words of Mackenzie: "All subjects that have attained the position of a science, have passed through stages of which the collection of facts was the first. So long as these facts were uncoordinated, their accumulation tended to confusion and there came to be recognized the need for an ordered arrangement based upon a law of Nature." If I have succeeded in bringing together under a common mechanism types of pain hitherto widely separated, I shall have simplified the subject and accomplished a little in the search for those governing principles which classify the details of our knowledge and make a science of our art.

Summary.

1. The viscera are insensitive to many stimuli, in spite of the opinions of Kast and Meltzer.

2. This insensibility has an interesting biological significance.

3. Sir James Mackenzie made an important step forward in the interpretation of pain by describing its reflex effects, but he erred in denying any sensibility to the viscera and he failed to describe the adequate stimulus which initiates the impulses in the reflex paths.

4. Lennander's work is most important in emphasizing the sensibility of the parietal peritoneum, but his theories fail when applied to purely splanchnic pains.

5. Hurst distinguished the referred and the truly visceral elements in splanchnic pain. He pointed out that the viscera may be sensitive if the adequate stimulus be employed, although they may be insensitive to other forms of interference. He considered distension to be the adequate stimulus, but failed to take into account the facts that extreme distension may cause no pain and that visceral pain may occur in the absence of any distension. I believe that the reaction of a hollow viscus to experimental or pathological distension depends upon its physiological habits. The stomach and the renal pelvis illustrate this principle from opposite standpoints.

6. The characteristics of pain in peptic ulcer are described, special stress being laid upon its usually steady nature. It is noted that hæmorrhage influences the pain.

7. Current theories (Mackenzie's, Lennander's, Hurst's, Ryle's and Carlson's)) are quite inadequate to explain the characteristics of ulcer pains.

8. Somatic pain may, of course, arise from mechanical interference. By dragging on the parietal peritoneum, prolapse of the viscera may cause pain which is really somatic.

9. The pain in all organic diseases of the splanchnopleure can be brought under a common mechanism with somatic pain. Compression of nerve fibres has been shown clinically and experimentally to be the essential common factor.

10. Compression is of two great varieties: (i) that due to vascular and cellular congestion in the tissues and (ii) that due to powerful muscular contraction.

11. Pains depending upon different mechanisms have definite and characteristic attributes, following naturally from the mode of pain production. This is a most important principle. Apart from pain due to mechanical interference with somatic tissues, the congestive and the peristaltic are the two great varieties of pain.

12. The steady nature of peptic ulcer pains suggests a steady cause, namely congestion. Further scrutiny of this conception provides for the peculiarities of ulcer pains a satisfactory explanation hitherto impossible. Pain is therefore pro-

duced in a chronic ulcer of the stomach in the same way as in a chronic ulcer anywhere else, for example in the leg.

13. Congestion requires a certain amount of rigidity in the tissues, in order that compression may be brought to a stage adequate for pain production. Congestion in rigid tissue is present in every case of chronic peptic ulcer. The essentials for pain production according to previous notions could be demonstrated in but a few of the patients.

14. The alkalis have been investigated radiographically and kymographically in the healthy and in the diseased subject. They have no effect on gastric peristalsis. Sodium bicarbonate causes a relaxation of the pyloro-duodenal musculature.

15. The pseudo-ulcer pains in appendix and gall bladder disease depend upon lymphangitis and lymphadenitis in the pyloro-duodenal region.

ACKNOWLEDGEMENTS.

It is with pleasure that I acknowledge my indebtedness to Mr. H. B. Devine, in whose clinic this work has been carried out. My thanks are due also to Professor Osborne, through whom I was able to obtain the apparatus for my kymographic study, and to Mr. Barnes, who gave me valuable assistance in the use of the apparatus.

APPENDIX.

CASE I.—Mr. W. D. O'B., *ætatis* thirty, complained of a feeling of epigastric fullness and wind coming on soon after meals for the last three years. He vomited very often in the afternoons and this gave relief proportionate to the amount vomited. Sometimes he noticed in the vomited matter food taken at breakfast. He suffered years ago from attacks of epigastric pain half an hour after meals, which lasted for some weeks and were separated by long intervals of freedom; they were never very severe. His stomach was visibly distended and there was no tender point. X ray examination revealed distinct pyloric obstruction with hyperperistalsis, confirmed later at operation. This patient illustrates modification of symptoms by stenosis.

CASE II.—Mr. D., *ætatis* seventy, complained of attacks of gnawing pain in the epigastrium aggravated at once or a few minutes after meals, relieved formerly by the doctor's alkaline medicine which now fails. The attacks are worse in cold weather and are separated by intervals of freedom, at one time lasting for six or twelve months, now only for a few weeks or months at most. A test meal revealed hyperchlorhydria, the free acid in his fasting contents and in three samples after a gruel meal being 40, 15, 65 and 70 cubic centimetres of decinormal hydrochloric acid in one hundred cubic centimetres of gastric contents. X ray examination showed the niche of a duodenal ulcer, which was found at operation to have invaded the pancreas and produced a large inflammatory tumour. This patient illustrates the modification of symptoms by penetration of neighbouring viscera by the ulcer.

CASE III.—Mr. O., *ætatis* forty-four, complained of epigastric pains and soreness for the last sixteen years, lately going through to the back. Food and alkalis relieved, but the period of relief is getting less; a bismuth mixture which was effective two and a half years ago, now fails entirely. Food used to relieve at once, now only after five or ten minutes. Eight years ago his medical advisor drained an abscess in the upper part of the abdomen due to perforation of a duodenal ulcer. X ray examination showed persistent irregularity of the pylorus and duodenum. Gastric contents aspirated at operation contained twenty-five cubic centimetres of decinormal free hydrochloric acid in one hundred cubic centimetres of gastric content. At operation an ulcer was found invading the pancreas, but it was not possible to tell whether it had originated in the stomach or in the duodenum. This patient illustrates the symptom modification by penetration of the ulcer.

CASE IV.—Miss T., *ætatis* forty-four, a trained nurse, gave a history of epigastric pains for twenty years, worse in winter and with intervals of freedom. The pain came half to one hour after meals and was worse to the left of the mid-line. It used to be relieved completely by alkalis and vomiting, but these gradually failed to relieve, so that twelve months ago she had given up taking sodium bicarbonate. Vomiting still gave slight relief. The stomach was seen radiographically to be orthotonic and undilated and peristalsis was normal. Operation disclosed an ulcer high up on the lesser curvature, non-penetrating, but adherent to surrounding structures including the pancreas and slightly to the anterior abdominal wall. This patient illustrates the modification of symptoms by adhesions.

CASE V.—Mr. R., *ætatis* sixty-two, the father of a medical man, was referred for operation for gastric carcinoma. For four months he had been getting epigastric pain and soreness worse on the right side, coming regularly at 11 a.m. and 4 p.m. and relieved by food and alkalis. He had lost one and a half stone in weight during the four months. His gastric contents had a low acidity, rising to twenty-seven cubic centimetres of decinormal hydrochloric acid in one hundred cubic centimetres of gastric contents after a gruel meal. Operation disclosed a small duodenal ulcer. This patient shows hypochlorhydria with typical ulcer pains and alkali relief.

CASE VI.—Mr. P., *ætatis* nineteen, had been complaining for two months and for several weeks during last summer of steady nagging epigastric pain, not severe as a rule. It lasted all day, becoming worse about one hour after each meal, but since undergoing medical treatment it appeared only after the midday meal. It is relieved, but not entirely, by the next meal, by alkalis and by vomiting which occurred occasionally. His epigastrium was tender on palpation. Repeated radiographic examination revealed definite deep tenderness in the pyloro-duodenal region, but the outline of the viscera was quite normal. There was also a zone of more superficial and less definite tenderness above the lesser curvature of the stomach. The benzidene test revealed no occult blood in the fæces. Fractional gastric analysis was performed twice, samples being withdrawn at half hourly intervals. The free acidity figures were (*a*) 0, 0, 5, 15 —, 0; (*b*) 0, 0, 15, 35, 35, 30. At operation no ulcer was found, but the appendix was obviously pathological, long and dilated, the seat of chronic inflammation. Removal of the appendix gave relief of symptoms and when the patient was heard of five months after operation, there had been no return of his trouble. This case history illustrates: (i) hunger pains depending on chronic appendicitis, (ii) the steady nature of these pains, (iii) their relief by soda although the gastric acidity was not high, (iv) the deep tenderness and the more superficial referred tenderness. It makes clear the mechanism of production of these pains, for the deep tenderness in the pyloro-duodenal region and the pains are probably both associated with a lymphangitis in the drainage area of the appendix.

CASE VII.—Mr. F.D., *ætatis* fifty, was suffering from jejunal ulcer following a gastro-enterostomy done three years ago in another State for duodenal ulcer. Before the operation he suffered from epigastric discomfort occurring regularly two hours after meals and he had had some severe hæmorrhages. After the operation he began to get epigastric pain, relieved by soda, magnesia and hot water, sometimes by meals, but not always. He had six hæmorrhages since the operation. Gastric analysis by Dr. F. L. Apperly, showed mere traces of free acid and very low total acid. Operation confirmed the diagnosis of jejunal ulcer. This patient illustrates pain relief by alkalis with a low or absent acidity.

CASE VIII.—Mr. W., *ætatis* thirty-seven, complained of a sinking feeling and epigastric pain particularly at 11 a.m., at 4 p.m. and at bedtime. These were relieved by food and by soda. The patient was a grocer and in the habit of taking a pinch from the soda tin to relieve symptoms. Fractional gastric analysis continued for two and a half hours, revealed no free acid, either before or after his

operation. Radiography showed the stomach to be hypotonic with a persistent mid-gastric spasm. At operation a tiny chronic ulcer of the lesser curvature with enlarged pyloric glands and a pathological lesion of the appendix were found. This case will illustrate the relief of hunger pains brought about by alkalis even in achlorhydria. When considered in conjunction with Case VI it serves to illustrate the causal relationship between chronic appendicitis and peptic ulcer; their clinical resemblance and the dependence of ulcer pains and pseudo-ulcer pains upon similar mechanisms can be appreciated.

REFERENCES.

(1) Sir James Mackenzie: "Symptoms and their Interpretation," 1920.

(2) K. G. Lennander: "Abdominal Pain, Especially Pain in Connection with Ileus," *The Journal of the American Medical Association*, September 7, 1907, page 836.

(3) S. J. Meltzer: Discussion on Ileus, *The Journal of the American Medical Association*, September 21, 1907, page 1013.

(4) J. F. Mitchell: *The Journal of the American Medical Association*, September 21, 1907, page 1015.

(5) A. J. Carlson and A. B. Luckhardt: "Studies on the Visceral Sensory Nervous System: V. Cardiac and Vasomotor Reflexes Induced by Visceral Stimulation in Amphibia and Reptilia," *American Journal of Physiology*, February 1, 1921, page 13.

(6) W. R. Meeker: "Splanchnic Anæsthesia: An Analysis of Forty-two Cases," *Archives of Surgery*, March, 1925, page 699.

(7) H. Head: "On Disturbances of Sensation, with Especial Reference to the Pain of Visceral Disease," *Brain*, 1893, Volume XVI, page 1.

(8) H. Finsterer: "Anæsthesia in Abdominal Surgery," *The British Medical Journal*, August 14, 1926, page 290.

(9) William Beaumont: "Experiments and Observations on the Gastric Juice and the Physiology of Digestion," 1833.

(10) W. A. Bastedo: "The Dilatation Test for Chronic Appendicitis," *The American Journal of the Medical Sciences*, July, 1911, page 11.

(11) A. F. Hurst: "Medical Essays and Addresses," 1924.

(12) A. F. Hurst: "The Sensibility of the Alimentary Canal."

(13) A. J. Walton: "A Text-book of the Surgical Dyspepsies," 1923.

(14) R. D. Carman: "The Roentgen Diagnosis of Diseases of the Alimentary Canal," 1921.

(15) L. Reynolds and C. W. McClure: "Motor Phenomena Occurring in Normal Stomachs and in the Presence of Peptic Ulcer and its Pains, as Observed Fluoroscopically," *Archives of Internal Medicine*, January, 1922, page 1.

(16) J. Homans: "The Relation of Pain in Gastric and Duodenal Ulcer to Muscular Activity of the Stomach," *The American Journal of the Medical Sciences*, January, 1919, page 74.

(17) L. J. Hardt and E. G. Joseph: "Kymographic Studies of Various Pathological Conditions of the Stomach," *The Medical Clinics of North America*, July, 1923, page 63.

(18) A. O. Wilensky: "A Study of the Motor Disturbances Accompanying Ulcer of the Stomach or Duodenum and the Changes Produced by Operation," *Annals of Surgery*, June, 1917, page 730.

(19) A. J. Carlson: "Contributions to the Physiology of the Stomach: XLIV. The Origin of the Epigastric Pains in Cases of Gastric and Duodenal Ulcer," *American Journal of Physiology*, December, 1917, page 81.

(20) B. G. A. Moynihan: "Some Problems of Gastric and Duodenal Ulcer," *The British Medical Journal*, February 10, 1923, page 221.

(21) J. Vandorfy: International Abstract of Surgery, *Surgery, Gynecology and Obstetrics*, October, 1925, page 274.

(22) H. B. Devine: "Early Diagnosis of Gastric Cancer," THE MEDICAL JOURNAL OF AUSTRALIA, August 14, 1926, page 209.

(23) H. B. Devine: "Basic Principles and Supreme Difficulties in Gastric Surgery," *Surgery, Gynecology and Obstetrics*, January, 1925, page 1.

(24) A. B. Luckhardt, H. T. Phillips and A. J. Carlson: "Contributions to the Physiology of the Stomach: LI. The Control of the Pylorus," *American Journal of Physiology*, October 1, 1919, page 57.

(25) M. McC. Baird, J. M. H. Campbell and J. R. Hern: "The Importance of Estimating Chlorides in the Fractional Test Meal Samples and Some Experiments with the Duodenal Tube," *Guy's Hospital Reports*, January, 1924, page 23.

(26) L. G. Cole: "Motor Phenomena of the Stomach and Cap Observed Roentgenographically," *American Journal of Physiology*, December, 1917, page 618.

(27) L. J. Hardt: "Pain in Active Pathologic Processes in the Stomach or Duodenum: Gastric and Duodenal Contractions as the Direct Cause," *The Journal of the American Medical Association*, March 23, 1918, page 837.

(28) A. J. Carlson: "Contributions to the Physiology of the Stomach: V. The Influence of Stimulation of the Gastric Mucosa on the Contractions of the Empty Stomach (Hunger Contractions) in Man," *American Journal of Physiology*, September, 1913, page 245.

(29) H. Ginsburg, W. Tumpowsky and I. Hamburger: "Contributions to the Physiology of the Stomach: XXXV. The Newer Interpretation of the Gastric Pain in Chronic Ulcer," *The Journal of the American Medical Association*, September 30, 1916, page 990.

(30) J. A. Ryle: "Visceral Pain and Referred Pain," *The Lancet*, May 1, 1926, page 895.

(31) W. M. Bayliss: "Principles of General Physiology," 1918.

(32) Boldyreff, quoted by F. T. Rogers and L. L. J. Hardt: "Contributions to the Physiology of the Stomach: XXVI. The Relation Between the Digestive Contractions of the Filled and the Hunger Contractions of the 'Empty' Stomach," *American Journal of Physiology*, August 1, 1915, page 274.

(33) W. B. Cannon and A. L. Washburn: "An Explanation of Hunger," *American Journal of Physiology*, March, 1912, page 441.

(34) A. J. Carlson: "Contributions to the Physiology of the Stomach: I. The Character of the Movements of the Empty Stomach in Man," *American Journal of Physiology*, December, 1912, page 151.

(35) A. J. Carlson: *Ibidem.*

(36) B. G. A. Moynihan: "The Diagnosis and Treatment of Duodenal Ulcer," *Proceedings of the Royal Society of Medicine*, January, 1910, Surgery Section, page 69.

(37) Howell's "Text-book of Physiology," 1923, page 629.

(38) B. G. A. Moynihan: "The Diagnosis and Treatment of Chronic Gastric Ulcer," *The British Medical Journal*, December 13, 1919, page 765.

(39) E. P. Poulton: *The Post-graduate Medical Journal*, May 8, 1926, page 105.

(40) W. H. Spencer, G. P. Meyer, M. E. Rehfuss and P. B. Hawk: "Gastro-intestinal Studies," *American Journal of Physiology*, February 1, 1916, page 459.

(41) J. C. Friedman: "Time Relations of Gastric Pains, with Special Reference to Gastric Adhesions," *The American Journal of Medical Sciences*, May, 1916, page 735.

(42) E. C. Rosenow: "Focal Infection and Elective Localization of Bacteria in Appendicitis, Ulcer of the Stomach, Cholecystitis and Pancreatitis," *Surgery, Gynecology and Obstetrics*, July, 1921, page 21.

(43) L. R. Braithwaite: "The Flow of Lymph from the Ileo-Caecal Angle and its Possible Bearing on the Cause of Duodenal and Gastric Ulcer," *British Journal of Surgery*, July, 1923, page 7.

(44) D. Bercenau: International Abstracts of Surgery, *Surgery, Gynecology and Obstetrics*, February, 1925, page 123.

(45) J. B. Deaver and D. B. Pfeiffer: "Chronic Pancreatitis," *Annals of Surgery*, June, 1914, page 841.

(45a) B. G. A. Moynihan: "Appendix Dyspepsia," *The British Medical Journal*, January 29, 1910, page 241.

(46) Ian Schoemaker: "The Red Stomach," *Surgery, Gynecology and Obstetrics*, March, 1925, page 305.

(47) H. L. Barnard: "Contributions to Abdominal Surgery," 1910.

(48) F. L. Apperly: "The Mechanism of Hyperchlorhydria," THE MEDICAL JOURNAL OF AUSTRALIA, January 13, 1923, page 33.

(49) F. L. Apperly: "Duodenal Regurgitation and the Control of the Pylorus," *The British Journal of Experimental Pathology*, 1926, Volume VII, page 111.
(50) F. L. Apperly: "The Use of Hydrochloric Acid and Sodium Bicarbonate in the Treatment of Certain Common Dyspepsias," THE MEDICAL JOURNAL OF AUSTRALIA, March 27, 1926, page 354.

Reports of Cases.

ACUTE NICOTINE POISONING.

By F. A. E. LAWES, M.B., Ch.M.,
Hornsby, New South Wales.

As nicotine poisoning is somewhat rare, a record of the following case is suggested.

Mrs. E.B., aged forty-six years, first consulted me on April 3, 1927, complaining of being very "run down" and in a constant state of nervous excitement. She said that her mind was never quiet, that she worried over trifles, was restless and slept badly. She was staying in this district with her mother and sister, having been a month away from her husband and two children. Although fond of them, she refused to see them again. She complained that her constant mental restlessness was wearing her out.

Her past history is interesting. At the time of her marriage, she was twenty-eight years of age and her husband thirty. For some reason she thought that she ought not to have any children and so had no intercourse with her husband for some time. Later she allowed this and had two children, a boy who is now seventeen years of age and a girl of thirteen. After the birth of her second child she had no more intercourse.

Her mother states that she was always a nervous girl. Six years ago she had an operation for replacement of the uterus. Two years ago the appendix was removed.

About two months before I saw her she had been under treatment for neurasthenia by a women's specialist. She went to his rooms and something was done to the *cervix uteri* which was said to be diseased and the cause of all her trouble. A cure was promised and she was in high hopes. The exact nature of the treatment was unknown to her, but she bled freely from the uterus after each treatment and continued to do so to such an extent that ergot was administered to control it. Her state of mind after all this was much worse.

Physical examination revealed a thin, but well preserved woman, very intelligent, but very excitable and talkative. Her reasoning power was good, also her memory. She talked a lot of her various troubles.

She had *pyorrhœa alveolaris* and bad teeth in the lower jaw. Her throat was healthy, as were her heart and lungs. The abdominal organs appeared to be normal. The uterus was felt to be in good position. The cervix was normal. The urine was normal. The pupil reactions and eye movements were normal. The knee jerks were very active. There were no other noteworthy features in the examination of the nervous system.

She was given a mixture of bromide, also "Luminal" at night. She noticed some slight improvement, but after expressed a wish to die and sometimes suggested suicide.

On April 12 Dr. Andrew Davidson saw her in consultation. He formed the opinion that her condition was one of hysteria, probably resulting from her unnatural life and rendered worse by her mouth condition and consequent digestive disorder. He advised hospital treatment, which was refused by the patient.

On April 22 at 8 a.m. her sister summoned me urgently saying that the patient had swallowed poison of unknown nature. When I arrived ten minutes later, she was dead. On inquiry it transpired that she had found a bottle labelled "poison" in the motor shed the day before and had secreted it in her room. She had taken some of the contents of this bottle just after her mother and sister had left the room. Her sister told me that within three minutes of leaving the patient's room she heard loud sounds of retching and rushed back to the room. She saw her sister making violent efforts at vomiting, but without result. She was in a sitting position in bed and was swaying from side to side. She admitted having taken poison and seemed quite clear mentally. An attempt was made to administer an emetic, but the teeth were tightly clenched. A few seconds later the spasm relaxed, but the patient could not swallow and the fluid ran out of her mouth. Then she sank back without a sound, a little dark coloured fluid with mucus came out of her mouth and nose and she was dead. There were no convulsions and she appeared not to suffer any pain.

The bottle was labelled "Black Leaf 40." It had been purchased at a florist's shop and was a 40% solution of nicotine sulphate. The stock gallon tin was labelled in this way. It is commonly used in a dilution of one in forty to one in eighty for spraying fruit trees.

In Taylor's "Medical Jurisprudence" two cases of acute nicotine poisoning are recorded. One was in Belgium in 1851, a case of homicide, and one in England in 1858, a case of suicide.

I am informed by the Government Analyst, Dr. Cooksey, that a case is reported from the City Coroner's Court in which death is recorded as due to nicotine poisoning. This was in September, 1926. Dr. Cooksey assures me that in his experience death by nicotine poisoning is very rare.

Analytical Department.

"CAPROKOL."

IN our issue of June 4, 1927, Dr. Veader Leonard took exception to a statement appearing in an article entitled: "A Retrospect" dealing with "Hexyl-Resorcin." We undertook at that time to subject this substance to bacteriological and pharmacological tests and to publish the results as soon as they were available. Samples of "Caprokol" were kindly placed at our disposal by the Sydney agent of the British Drug Houses, Limited. The samples were sent to a clinician and a bacteriologist, neither of whom had any experience of the drug. In the course of six months five patients were carefully treated with "Caprokol" and the results were checked by the making of cultures from the urine.

The first patient was a female who had suffered from chronic pyelitis for about two years. After "Caprokol" had been administered in accordance with directions a heavy growth of *Bacillus coli communis* was obtained. The number of colonies per cubic centimetre of urine increased during the six weeks of treatment; no other drug was used.

The second patient was a male with a *cauda equina* lesion. He had incontinence of urine and chronic cystitis. The urine was cultured and a moderately heavy growth of *Bacillus coli communis* was obtained. The cystitis cleared up to some extent under the influence of "Caprokol" and of washing out the bladder. At the end of several weeks there was still a growth of *Bacillus coli communis* in the urine.

The third patient was a female with chronic pyelitis of several months' duration. A moderate growth of *Bacillus coli communis* was obtained from the urine. "Caprokol" was exhibited for some weeks, but the number of colonies per cubic centimetre of urine remained practically unchanged.

The fourth patient was also a woman. She had had chronic pyelitis for about eighteen months. Culture of the urine yielded a moderate growth of *Bacillus coli communis*. Although some diminution in the number of colonies per cubic centimetre of urine was noted while "Caprokol" was being administered, there was still an ample growth of *Bacillus coli communis* during the whole period.

The fifth and last patient was a female who had had chronic pyelitis for some months. There was a heavy growth of *Bacillus coli communis* on culturing the urine. "Caprokol" was given for several weeks, but the number of colonies did not become lessened.

These results appear to justify the conclusion arrived at in the article that "Hexyl-Resorcin" has been condemned by many urologists. It is obviously of smaller therapeutic value than "Hexamine" and is not to be recommended.

The Medical Journal of Australia

SATURDAY, JANUARY 21, 1928.

A Retrospect.

Gynæcology.

THERE has been an unusually large amount of excellent work on gynæcological subjects published by Australian investigators. The first place belongs to F. A. Maguire, whose monumental article in the pages of this journal represents the result of his painstaking, skilful and meticulously accurate study of the anatomy of the female pelvis. He has based his record on his careful observations of no less than fifteen pelves, some of which were dissected by himself. Various methods of making sections were employed. Maguire has resisted the temptation to engage in speculation; he has recorded unchallengeable facts and on this account his work will remain authoritative.

In the realm of physiology Wilfred Shaw, of London, has reported further investigations into the histology and functions of the ovary with special reference to the interstitial cells. He has extended our knowledge of ovulation. Beckwith Whitehouse has studied the functional changes of the *corpus luteum*. He has arrived at the conclusion that menstruation is the monthly abortion of an unfertilized ovum and the menstrual discharge is the lochia of this abortion. His investigations have led him to reconcile the structural changes with the cycle of events in the human female.

In connexion with inflammatory conditions mention should be made of H. H. Schlink's work on pelvic lymphangitis, reported to the second session of the Australasian Medical Congress (British Medical Association). He has been able to prove that gonococci can penetrate the basement membrane of the racemose glands and wander into the subperitoneal tissue, separated from the mucosal lumen by the whole thickness of the muscular coat. His work on the lymphatic spread of infection along the lymphatic paths with its definite pictorial evidence will find recognition throughout the world.

Malignant disease of the pelvic organs has been investigated from many points of view and much useful information has been forthcoming by the reports of single observations. W. D. Saltau has collected the records at the Women's Hospital, Melbourne, of women treated by radium during the past five years. He has found that radium undoubtedly exercises a beneficial effect on malignant growths whether applied before operation or after it. Very few patients with inoperable neoplasms were cured by radium, although this treatment has a palliative action. Reginald Davies has recorded the details of an endometrioma of the posterior wall of the uterus in a woman aged forty-one. The growth had invaded both ovaries and the peritoneum of the pouch of Douglas. He pointed out that the several hypotheses that have been put forward to explain the wandering of the endometrium fail to satisfy the conditions discovered in his case.

Some useful studies have been carried out on the prognosis in the various forms of malignant disease of the uterus and cervix. The American figures reveal that approximately one-half of patients with spinal cell cancer of the cervix remain well throughout a five year period, while under 10% survive for five years when affected by spindle cell cancer. The striking difference in malignancy between malignant adenomata of the uterus and adeno-carcinoma has been strongly emphasized.

F. A. Maguire has given details of a remarkable malignant condition treated in a most courageous manner. The bladder and urethra were extirpated and the ureters transplanted into the pelvic colon. The condition appeared at first sight to be beyond the aid of surgery.

Cecil Coglan has reviewed the modern work on the causation of sterility in women. He has found that the sterility rate in Australia is lower than it is in other countries. A clear account is given of the structural and functional defects leading to sterility.

Obstetrics.

The art and science of obstetrics do not develope *pari passu* with the rest of medicine, because the process of childbirth is a physiological one and

under normal conditions good midwifery means watching and waiting. The obstetrician has to be versed in sound principles of asepsis and he must be prepared to prevent abnormalities and irregularities rather than to meet them when they arise.

R. Marshall Allan published an interim report on the maternal mortality and morbidity in Victoria. As Director of Obstetrical Research, he has had the task of investigating the position in Victoria and of initiating those reforms that may be needed to reduce the number of deaths and the amount of illness arising from childbirth. His report is an excellent summary of the problems that must be solved in every part of the world.

A. E. R. White and J. C. Windeyer have dealt with puerperal sepsis. The former has analysed the experience at the Women's Hospital, Melbourne, and has drawn conclusions after full consideration of the clinical course of the fever after childbirth or abortion and of the bacteriology of the conditions. He finds that it is essential that an early diagnosis of septicæmia be made, if treatment is to yield good results. The diagnosis can be made only if a blood culture is undertaken with the blood of every parturient woman and every woman after abortion who has pronounced fever for over twenty-four hours. It has been found that good results can be obtained with mercurochrome. J. C. Windeyer delivered the Joseph Bancroft Memorial Lecture in Brisbane. After weighing all the facts ascertainable from the records of the Royal Hospital for Women, Sydney, he formed the opinion that three things were needed for the prevention of puerperal infections. The patients should be carefully watched and handled during the whole of the pregnancy; when an abnormal condition is discovered, the patient should be treated in hospital; a simple and efficient technique should be adopted by the obstetricians in every labour.

The causes of death from infection following abortion have been investigated by R. Magarey, J. B. Cleland and J. C. Sleeman. The infections were caused by *Bacillus welchii* and were characterized by gas formation in the uterus and jaundice.

John H. Teacher has investigated the question of the involution of the uterus after labour. He has followed the process of normal involution and has determined the degree of abnormality that does not interfere with the regular changes. He has defined each stage from the point of view of time and has shown how pathological processes affect the normal course of events.

Doris Gordon, speaking at the Australasian Medical Congress (British Medical Association) at Dunedin, described her experience and made out an excellent case for the defence of the careful, conscientious general practitioner in connexion with the incidence of puerperal sepsis.

The association of mental disorder with pregnancy, the puerperium and lactation has been considered by R. S. Ellery who has performed a valuable service in helping to remove one of the many myths which were introduced into medical doctrines at some unknown time and in some unknown manner. These fabulous conceptions are difficult to eradicate from the book of knowledge and remain as a reproach to the intelligence of the teachers of medicine. Ellery shows in an unassailable way that there is no such thing as a puerperal insanity. The puerperium with its attendant disturbances of the habitual functions of the body may influence the outward manifestations of a psychosis or may even precipitate an attack, but the insanity is not caused by this physiological process and should not be regarded as puerperal or lactational. In close connexion with this work is that of S. Evan Jones, S. J. Minogue, Constance E. D'Arcy and D. Macomber. The two first mentioned psychiatrists have examined the subject of the mental disorders encountered in women at the time of the climacteric, Constance D'Arcy has given an admirable account of the nervous disorders observed after the natural and artificial menopause and Macomber has dealt with the emotional changes traceable to the sexual life of women. The whole subject is a complex one. While Evan Jones and Minogue have left no doubt concerning the falseness of the doctrine that the end of sexual life in a woman is a cause of insanity and have shown that the association of the menopause with mental disorder is far less frequent than it is believed to be, the balance of the organs of

internal secretion is modified when one or more of these organs become inactive and a nervous instability manifests itself in women whose mentality is not vigorous, until a new balance is attained. Moreover, it has been shown with exemplary clarity that sexual activity in its manifold forms is correlated in the most intimate manner with emotional states and that it is probably not in accordance with fact to assume that the use or abuse of sexual function has no ætiological relationship with emotional disorders.

Some interesting observations have been made on the significance of acute appendicitis arising in the course of pregnancy by R. A. Wilson. This may be regarded as a specific instance of the general doctrine that obstetrics are or should be preventive medicine. The way to protect the pregnant woman from the grave dangers associated with this combination is to deal with the diseased appendix before pregnancy occurs.

At the Congress held in February in Dunedin, A. M. Wilson and H. Jellett discussed the perennial problem of the indications for Cæsarean section and are to be congratulated on the convincing manner in which they have justified their teaching. If their advice were accepted throughout Australasia, the harm done by the abuse of a valuable surgical procedure would disappear.

Therapeutics.

Pharmacology represents the least exact of the branches of medical science. With but few exceptions drugs given in so-called therapeutic doses produce so little effect on the body that the improvement ascribed to their use in pathological processes must be caused either by the effect of suggestion or by some circumstance attending the treatment of the disease. The British Pharmacopœia is full of details concerning the preparation of a very large number of medicaments and added to this the pharmacist and the manufacturing chemist are producing new drugs at a disconcerting rate. It is extremely doubtful whether the human race would be the poorer if this unmeasured traffic were discontinued and the medical profession were required to confine its therapeutics to a dozen well tried drugs.

The year 1927 has witnessed the introduction of a heavy load of new preparations. We know of none of assured value. Last year reference was made to "Hexyl-Resorcin," a substitutional preparation for the old "Urotropine." At the instigation of this journal tests have been carried out in an objective manner and it has been found that this preparation, which is known by the trade name of "Caprocol" has a very limited action in reducing the *Bacillus coli communis* content of the urine in cystitis and pyelitis.

Some sound work has been carried out in connexion with calcium metabolism and calcium medication. A. T. Cameron has studied the calcium content of normal and abnormal tissues, including bone, blood serum and muscles. He has estimated the daily calcium requirement of the healthy child and adult and the calcium loss. Since the parathyreoid probably controls calcium metabolism, it is not surprising to find that this element is deficient in tetany. The calcium content of the blood is diminished in pneumonia and in nephritis. Inorganic calcium salts appear to influence to some extent the excessive loss of calcium associated with some diseases.

The therapeutic value of iodine in the prevention and treatment of goître was discussed by the New Zealand authorities at the second session of the Australasian Medical Congress (British Medical Association), Dunedin, 1927. C. E. Hercus has correlated the iodine intake of persons in goîtrous and non-goîtrous districts and has taken into consideration the iodine content of the soil. He has suggested that the use of iodized salt for cooking and table purposes should be made compulsory in all goîtrous areas.

New problems continue to arise in connexion with the administration of "Insulin" for diabetes. R. D. Lawrence and O. B. Buckley have endeavoured to discover the cause of the failure of "Insulin" to control the blood sugar level in the presence of certain infections. They had postulated a thyreo-adrenal mechanism which is directly antagonistic to the insulin mechanism of the pancreas. It has been shown that fever and some infections influence the thyreo-adrenal apparatus and interfere with the regulation of the sugar metabolism. The storage

of glycogen in the liver and in muscles is effected under the stimulation of a carbohydrate intake and a raising of the blood sugar level. The thyreo-adrenal mechanism, particularly if increased by infections, mobilizes the sugar. If this action is excessive, it may be impossible for "Insulin" to counteract this process. While this explanation is speculative, it is an indication of a lack of definite knowledge in regard to the action of "Insulin." Clinicians have been embarrassed by the fact that when their patients have been taking large doses of "Insulin," the advent of an intercurrent infection may render the "Insulin" inert and coma may be precipitated as a result.

G. Bruton Sweet and H. Graham Robertson have described their experience of the application of ultra-violet rays in the treatment of erythrœdema. A. J. Wood is not yet convinced that this treatment is curative, but he admits that he has not used it often enough. H. G. Robertson, like the workers in Europe, Gauvain, Rollier and Pacini, has found ultra-violet ray treatment of use in supplementing other treatment in infections of various kinds.

S. O. Cowen has discussed a large number of new remedies and has compared the action of various members of several groups. He has called attention to the fact that the proportion of worthless preparations among those widely advertised is great. K. McLean has reviewed the new antisyphilitic remedies and offers useful guidance in the choice among the bismuth and arsenic drugs. There is a general tendency among those who specialize in the treatment of syphilis, to rely on arsenic and bismuth and to restrict the use of mercury to a very great extent. On the other hand "Novarsurol" is well spoken of by J. C. Hossack and others as a diuretic in cardiac dropsy. It is an organic combination of mercury with diethylbarbituric acid. The literature of the year contains many favourable accounts of "Mercurochrome 220" injected intravenously for septicæmia.

Morphology.

The activities of anatomists in all parts of the world have been directed during the past twelve months and more to the elucidation of the yet unsolved problem of the double innervation of striated muscle and of the innervation of the alimentary canal. Boeke and Kuntz have published results of further research on the innervation of striated muscle. The evidence put forward by them confirms their earlier conclusions that endings of sympathetic nerves are found in striped muscle fibres which are already supplied with ordinary somatic endings. Boeke, however, has been unable to confirm the physiological results obtained by Royle and Hunter on goats. Royle has produced further convincing proof of his original findings.

In regard to the innervation of the gut, Kuntz and Hill have made valuable contributions which appear to bring the problem nearer to a solution. Reference was made in this place last year to the work of H. H. Woollard on the innervation of the heart. Since then Tudor Jones has made further additions to the scant knowledge that has been gathered on this difficult subject.

Investigations on the histogenesis of the ganglia of the sympathetic chain have led to the thesis that the neurones in these ganglia arise from cells of the medullary organ. This conclusion is in accord with the fundamental principle of differentiation of nervous tissue.

Consequent upon his researches into the innervation of muscle H. J. Wilkinson was led to undertake an interesting study of the pupillary reactions. He has brought forward an explanation of the phenomenon of the Argyll-Robertson pupil which fits in more fully with all the facts than any hypothesis suggested since the phenomenon was first observed sixty years ago.

G. H. S. Lightoller and A. N. Burkitt have published the final part of their work on the facial muscles of Australian aborigines. This work will be regarded as the standard for all future investigations on the subject. J. L. Shellshear has continued his valuable reports on the principles underlying the distribution of the arteries of the body, especially of the brain. He has demonstrated that the arterial supply of the cerebral cortex is precise in its distribution. He suggests that this might be used as an auxiliary to other methods of determining cerebral localization. He has further claimed that the precision of the arterial supply of the cerebral cortex adds confirmation to the law of arterial distribution enunciated by John Hilton.

C. W. Stump's work on the histogenesis of blood cells deserves special mention. He has adduced definite evidence in support of his contentions on the process of myelogenesis and in consequence he has advanced the study of hæmatology.

F. S. Hammett's work represents the result of an attempt to evaluate the part played by the thyreoid apparatus in the differential growth of mammals. R. O. Moody has carried out some important work on the position of the abdominal viscera. His conclusions are that enteroptosis, gastroptosis and coloptosis seldom if ever exist as pathological conditions. In view of the fact that these conditions have been freely discussed in medical literature during the past twenty-five years and that there is still much difference of opinion as to the exact significance of the terms, his conclusions are very interesting. Reference should be made to a paper read at the second session of the Australasian Medical Congress (British Medical Association), Dunedin, by J. Renfrew White, in the course of which the speaker showed that visceroptosis is not wholly or primarily an abdominal condition and that it is the result of postural defects rather than pathological processes. In effect the contributions will help to remove another vexed subject to the realms of medical mythology.

Radiology.

The term radiology is taken to include both the application of Röntgen rays in diagnosis and irradiation for treatment purposes. It is, however, more convenient to reserve under this caption treatment with radium and Röntgen rays and to include under the heading physical therapy other forms of irradiation such as actino-therapy, ultra-violet radiation, diathermy and other forms of heat ray applications, infra-red radiation and so forth.

In the realm of radiography the year 1927 has witnessed no new discovery, but the extension of many methods. The Graham test for gall-bladder disease has found wide application in all countries. Graham advocated the injection of the bromide salt of tetra-iodo-phenolphthalein. It has been found that the sodium salt, having a higher atomic weight, can be given in smaller quantities without reducing the physical effect. W. H. Stewart, C. G. Edwards and Anderson Stuart have favoured the oral method of application. It is simpler, perhaps not as accurate as far as dose is concerned, but sufficiently accurate for the purpose and just as reliable for radioscopical purposes. Cholecystography must be regarded as a great advance in gall-bladder diagnosis. Some excellent skiagrams have been reproduced in this journal. The subject of the radiographic diagnosis of duodenal lesions was fully discussed at the second session of the Australasian Medical Congress (British Medical Association), Dunedin, 1927. H. R. Sear demonstrated in his paper the fine degree of accuracy which an expert can attain in this class of work. At the same session D. F. Myers dealt in an able manner with the radiographic examination of the colon and H. F. Praagst discussed the subject of the reflex dyspepsias.

"Lipiodol" is being used extensively not only for the diagnosis of spinal tumours and obstructions in the spinal theca, but also in bronchiectasis. Q. U. Newell has employed "Iodipin" is gynæcological conditions on the same principle. J. F. Mackeddie has given an excellent account of the value of "Lipiodol" as a diagnostic agent. The fears concerning its possible toxicity would seem to be theoretical rather than practical.

Improvements in radiography have been brought about by the introduction of "Agfa" films, the large Müller tubes and the "Metallix" tubes, the 100 milliampère radiator Coolidge tube and various other devices. Many workers are using as much as 350 milliampères of current through the larger tubes, thus obtaining fine details with short exposures.

There has been somewhat of a lull in the advocacy of deep X ray therapy for malignant disease. This is not due to a diminution of enthusiasm on the part of the radiologists who claim to have obtained good results in this way, but rather to the fact that no new information has been produced. It would seem as though the acceptance of radiological treatment of malignant disease will be a gradual process, not a revolution. Douglas Quick has shown that with proper technique and dosage radium is capable of improving the prognosis of carcinoma of the tongue in a manner impossible by surgery. Other forms of cancer have yielded to X ray treatment in the hands of the leading radiologists.

Abstracts from Current Medical Literature.

DERMATOLOGY.

The New Method of Epilation in Tinea Tonsurans.

J. DEVANE (*The Medical Press and Circular*, August 3, 1927) gives a report on forty-five cases of epilation by thallium acetate. Although tonic symptoms have occurred in the experience of some dermatologists, the author observed no ill effects in his series of cases following the administration of thallium. The dose advised is eight milligrammes per kilogram of the body weight. Three injunctions should be strictly observed. The body weight, nude, should be accurately obtained. A standard reliable preparation should be used and it should be ascertained that the kidneys are functioning correctly. The required dose was ground into a fine powder and administered in a little hot water, no food being given for four hours. It is presumed that the drug acts through the endocrine and sympathetic systems. The hairs become loose about the sixth to eighth day. From the sixteenth to the nineteenth day the whole head should be bald. The hair grows again after four weeks.

The Ætiology of Eczema.

J. F. BURGESS (*Archives of Dermatology and Syphilology*, August, 1927) discusses the relationship of eczema to intestinal disturbances. In a series of patients there was considerable improvement following the use of intestinal therapy, such as saline purges, mild doses of mercuric chloride and *Bacillus acidophilus* culture. All these patients had a definite indicanuria, which disappeared as the condition improved. Bassler's modification of Jaffé's indican test was used. With the object of investigating the mechanism of the relationship, certain obtainable intestinal end-products and putrafactive bodies were tested in a series of eczematous and normal people. The substances used may be classified into two divisions, the amino-acid and the ptomaine bases. The substances were made up in 1 : 1,000 aqueous dilutions and the scratch method was applied. Of one hundred and nine persons suffering from eczema 16·6% yielded reactions chiefly to products of intestinal putrefaction. One hundred and two persons with other skin affections were tested and 2% reacted. All except two of the total number of two hundred and eleven persons tested gave a reaction to histamine.

Progressive Pigmentary Dermatosis.

H. J. TEMPLETON (*Archives of Dermatology and Syphilology*, August, 1927) reports two further cases of Schamberg's disease with a review of the literature on the subject. The pigmentary dermatosis occurred in young men, in one as a patch on the left thigh and in the other occurring on both ankles. In the differential diagnosis he mentions *angioma serpiginosum* and *purpura annularis telangiectodes*. The microscopic picture of Schamberg's disease distinctly differentiates it from the other two conditions mentioned. The writer states that mention has not been made in previous articles of the subject of treatment. In one of the cases described he applied carbon dioxide snow for twenty seconds, with the use of moderate pressure. After two applications with an interval of two weeks much improvement was obtained.

Parapsoriasis.

J. M. H. MACLEOD (*The British Journal of Dermatology and Syphilis*, October, 1927) reports a case of parapsoriasis in a pastry cook thirty-three years of age. There was no itching. The patches were a pinkish-fawn tint and varied in size from that of a sixpenny piece up to seven and a half centimetres in diameter. They were uninfluenced by any form of local treatment. The skin affection was associated with vasomotor rhinitis and with each attack of the latter the patches became inflamed. No septic foci were detected. The patient, owing to his occupation, was exposed a great deal to heat, a fact which has been noted in the histories of other patients.

Pemphigus.

L. HOLLANDER (*The British Journal of Dermatology and Syphilis*, November, 1927) describes the results of treating six patients with whole-blood transfusions for pemphigus. The patient's blood is typed into one of the four groups and direct blood transfusion is carried out at intervals of one week and repeated three to six times. Five hundred to six hundred cubic centimetres of blood are used. During the procedure the administration is discontinued and 0·6 mil of 1‰ adrenalin chloride is injected intradermally, when a tingling sensation in the toes and fingers, numbness of the extremities, palpitation, air-hunger or cyanosis is noted. Much improvement occurred in the general and skin condition of five of the patients. At the time of writing several were able to get about. The sixth patient died.

The Gold Treatment of Lupus.

H. H. WHITEHOUSE AND P. E. BECHET (*Archives of Dermatology and Syphilology*, November, 1927) discuss the treatment of certain dermatoses by the use of gold compounds with special reference to *lupus erythematosis*. Gold and sodium thiosulphate was the preparation used for all the patients with the exception of two, for whom gold protein was employed. The compound was injected intravenously once or twice a week in doses of one decigramme diluted in from two to five cubic centimetres of distilled water. Thirty persons with *lupus erythematosis discoides* were treated and only one did not show improvement. Four were completely cured and the majority of the remainder showed considerable improvement. One patient with *lupus vulgaris* did not benefit by the treatment. Several tuberculides treated responded rapidly. Reactions were rare and not serious except when the gold protein was used. The toxic symptoms in connexion with the latter were probably due to the protein element. The authors in their four months' experience regard the results as most favourable in a condition that is unusually intractable.

Calcium and Urticaria.

S. S. GREENBAUM (*Archives of Dermatology and Syphilology*, November, 1927) states that the reports in the literature on the calcium content and the coagulability of the blood in urticaria and allied conditions are meagre and contradictory. The estimations of calcium in the blood serum were made on sixty-three patients with all forms of urticaria. In all but one there was either a normal or increased calcium content. In half the number the coagulability was tested and a decrease was not found in any instance. Thus it is concluded that there is no scientific basis for the administration of calcium salts in urticaria. The writer also studied the effect of "Ephedrine," recently introduced into American medicine, in the treatment of this condition. The value of "Epinephrine" in the control of the subjective symptoms has previously been noted. In subacute urticaria "Ephidrine" is claimed to be a valuable adjunct to "Epinephrine," especially when the patient's tolerance to the latter is low.

Raynaud's Disease.

D. E. H. CLEVELAND (*Archives of Dermatology and Syphilology*, November, 1927) reports two cases of Raynaud's disease associated with other dermatoses. The disease developed during the course of *dermatitis herpetiformis* in a man aged forty years and was associated with *lupus erythematosis* in a woman aged sixty-eight years. The writer suggests that the toxæmia producing the dermatosis was responsible for the vasomotor derangement. He states that urticaria and scleroderma have been reported as being observed in connexion with Raynaud's disease.

UROLOGY.

Renal Amœbiasis.

M. SEGUROLA (*Vida Nueva*, July 15, 1927) thinks that the case of renal amœbiasis described by him is of in-

terest because it proves the existence of an amœbic invasion of the system in which it is impossible to determine a primary focus in the gastro-intestinal tract. The patient was a sailor who was admitted to hospital suffering from an influenzal attack. His urine contained traces of albumin, red cells and leucocytes, cells from the renal pelvis and a large number of histolytic amœbæ in both cystic and vegetative stages. On cystoscopic examination the bladder was found to be congested and inflamed. Ureteral catheterization was practised on both sides and the contents of the seminal vesicles and prostate were expressed. *Amœba histolytica* was found in all these specimens. The patient gave no history of gastro-intestinal disorders and repeated examination of the fæces failed to reveal the presence of amœbæ. The renal pelvis of each side was washed out with a weak solution of ipecacuanha and eight daily injections of six centigrammes of emetine were given. At the end of the course no trace of amœbæ could be found in the urine or in the prostatic or vesicular secretions. The writer believes that this is the first recorded case in which the amœba has been found in urine drawn directly from the kidney. He can find no record of the previous use of the ipecacuanha solution for lavage of the renal pelvis. In view of the absence of gastro-intestinal symptoms and of the fact that the patient entered hospital with a frank bronchial infection. Segurola thinks that the port of entry may have been the respiratory tract.

Leucoplasia of the Renal Pelvis.

Two cases of leucoplasia of the renal pelvis are reported by G. PEDROSO and P. G. LEQUERICA (*Archives de la sociedad de Estudios Clinicos de la Habana*, June, 1927). This brings the list of recorded cases up to forty-nine. The most characteristic symptom for differential purposes consists in the expulsion in the urine of epithelial masses desquamated from the renal pelvic surface. Other symptoms are due to concomitant disorders of the urinary tract. They include ureteral colic, hæmaturia, polyuria, pyuria and disturbances of micturition. Unless the expelled epithelial masses are recognized, the diagnosis is usually made at operation or *post mortem*. Two theories of origin are given. The first is that it is a metaplasia, that is to say, a transformation of one type of epithelium to a different type, the transformation being brought about by a chronic inflammation or irritation. The second is that it is attributed to a congenital condition. It has been pointed out in support of this hypothesis that at a certain period in the development of the embryo the primitive ureter, the future canal of Wolff, is in close relation for a certain time with the ectoderm. The authors are inclined to the belief that two factors are concerned, the embryological, in virtue of which implantation of ectodermal cells into the renal pelvis takes place, and chronic irritation or infection which induces proliferation of these cells and an invasion of the rest of the pelvis. The condition in one patient diagnosed by the appearance of the epithelial membranes in the urine, is said to have been cured by bathing of the renal pelvis with 5% silver nitrate solution. In all other instances surgical treatment, usually extirpation of the kidney, has been employed.

The Treatment of Prostatic Hypertrophy.

H. HABERER (*Wiener Medizinische Wochenschrift*, August 27, 1927) draws attention to the value of the retention catheter in cases of prostatic hypertrophy in enfeebled elderly men. He considers that patients with retention of urine should first be treated with a catheter tied in for a considerable period before operation is performed. In addition he advocates double vasectomy which will often obviate epididymitis after a prostatic operation. Treatment by vasectomy and the use of a retention catheter has at times enabled patients to urinate normally. In this way prostatectomy with its attendant risks was evaded.

Ureteral Transplantation in Bladder Carcinoma.

A. L. CHUTE (*Journal of the American Medical Association*, November 13, 1926) points out that since metastases form very late in bladder carcinoma, good results should be obtained from wide resection or from total cystectomy. He is astonished to find that no demonstrable metastases are found at autopsy on persons who have died of extensive malignant tumours of the bladder when death has been due to renal sepsis or to intercurrent disease. Among twenty-seven persons with bladder carcinoma metastases were discovered after death in only three. Local recurrence is to be feared unless the resection has been very wide and in tumours involving any part of the trigone total cystectomy is the only effective operation. Cystectomy has been very little used, partly because of technical difficulties, but chiefly on account of the preliminary difficulty of dealing in a satisfactory manner with the ureters. Lumbar nephrostomy or abdominal ureterostomy leaves very much to be desired from the patient's point of view, but the improved technique of implantation of the ureters into the sigmoid colon devised by Coffey gives some hope for the future. With this technique the ureter is included for about 2·5 centimetres within the wall of the sigmoid between its muscular and mucous coats, before it is made to enter the lumen. On the valve principle the intraintestinal pressure tends to keep the ureter closed. This pressure will not always prevent the entrance of bacteria into the ureter and some degree of pyelitis is to be expected. A recent important addition to the technique has rendered possible the implantation of the two ureters at the one time. This addition is the introduction of a number 12 French rubber tube, the upper end of which is inserted into the ureter to a point a little beyond that at which the canal enters the wall of the sigmoid, while the lower end is passed into the bowel and brought out through the anus. The tube prevents obstructive anuria due to post-operative œdema and this renders it safe to implant the two ureters at one time. In regard to the removal of the bladder it is wise to separate the prostate and seminal vesicles by perineal operation and some days later to open the abdomen and complete the whole prostato-cystectomy extraperitoneally as far as is possible.

A New Fulguration Cystoscope.

O. S. LOWSLEY (*Journal of Urology*, July, 1927) describes a specially designed cystoscope, the sheath of which is made of hard rubber, lined on the inside with a thin sheath of metal. The latter is in direct connexion with the tip of the beak of the instrument. This tip is made of metal and acts as a fulguration point. The metal tip is within the field of vision all the time the instrument is in use by reason of the fact that the lens system (McCarthy type) is directed forward. The telescope and light are both contained within the shaft of the instrument, thus allowing the greatest possible mobility in the conduct of intravesical fulguration. Continuous irrigation through the fenestrum of the instrument is possible; this insures a clear field of vision at the point of application of the electrode even in the presence of severe hæmorrhage.

Chronic Prostatitis.

W. S. PUGH (*Medical Journal and Record*, January 19, 1927) believes that in over 90% of cases chronic prostatitis is post-gonorrhœal. The gonococcus itself, however, can hardly ever be grown in cultures in this condition. Five hundred patients with chronic prostatitis were examined and the bacteriological findings were as follows: Staphylococci of various kinds, 22%; streptococci, 2%; *Bacillus coli*, 34%; *Bacillus pyocyaneus*, 1%; diphtheroids, 7%; *Bacillus proteus*, 1%; *Micrococcus catarrhalis*, 2%; sterile cultures, 37%. The relative incidence of the commoner symptoms is as follows: Itching or pain in the perineum, 83%; lumbar or lumbosacral pain, 90%; lowered sexual power, 15%; testicular pain, 23%; premature ejaculation, 6%; feeling of urethral fullness, 5%. Cysto-urethroscopy in chronic prostatitis often reveals polypoid masses at the bladder neck and in the posterior portion of the urethra; these masses should be removed by fulguration.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE WESTERN AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Perth Public Hospital on September 21, 1927, DR. A. H. GIBSON, the President, in the chair.

Anæsthetics.

MR. F. A. HADLEY said that he felt he was in a curious position opening up a discussion on anæsthetics, but if it were not for the surgeons, anæsthetists would not be required. Mr. Hadley discussed the needs of the surgeon and said that it was the aim of the anæsthetist to supply those needs. It was essential that the patient should feel no pain, either consciously or in the form of shock. The surgeon also required the necessary degree of relaxation which would allow him to perform his task with speed and with as little manipulation as possible. The surgeon was also interested in the after effects to the patient in that there should not be an excessive amount of salivation and that the vomiting should be eliminated as far as possible. Mr. Hadley expressed the hope that all forms of anæsthesia should be discussed during the evening and that the discussion would not be limited to any one point of interest.

DR. KASNER MOSS then read a short paper and demonstrated the method of intratracheal administration of ether for anæsthesia.

DR. G. R. TROUP demonstrated the nitrous oxide and oxygen method of anæsthesia.

DR. HORNER NELSON said that the first necessity of anæsthesia was that the patient should experience no pain, as Mr. Hadley had suggested. Dr. Nelson pointed out that deeper anæsthesia was necessary when the surgeon was exploring the abdomen than when engaged upon any particular part of it. Shock occurred in quite simple operations such as cutting an anal fissure and experience was needed to tell when it was most likely to occur. Relaxation was absolutely necessary and he did not recommend a large fixed retractor to overcome the lack of relaxation. In Dr. Nelson's opinion the anæsthetist should be well away from the surgeon—out of his road—but the screen which used to be used to separate them was no longer seen. With the idea of lessening the mucus, atropine was much in use, but Dr. Nelson did not administer it to all his patients. It was desirable that the head should be in a position of comfort and that the face should not be heavily covered with towels. Dr. Nelson said that he did not use more than four layers of gauze on the mask when administering ether by the open method and removed the towels when the patient was under the anæsthetic. The speaker said that all anæsthetists hoped that their patients did not have excessive vomiting. He believed that chloroform was badly given and realizing what a fearful thing "death on the table" was, he looked back in amazement to the days when chloroform alone was given.

DR. J. E. FERGUSON STEWART said that he had been giving chloroform for thirty years and liked it as an anæsthetic. It was handy to carry round, whereas the newer methods were expensive and difficult to transport. A man trained in a Scottish school knew how to give chloroform. Sir William Macewan had drilled into them that chloroform should be watched and if watched was safe. He had always been taught to listen to the breathing and not to watch the movements of the abdominal wall long after the breathing has ceased. The anæsthetist giving chloroform must hear the air go in. Dr. Stewart admitted that chloroform had a drawback in the amount of subsequent vomiting, but it did not set up the respiratory difficulties that ether did. Dr. Stewart said that he liked the nitrous oxide and oxygen method which Dr. Troup had described for minor operations.

DR. J. J. HOLLAND said that the country men would never be able to take advantage of the new methods such as the intratracheal anæsthesia, unless Henry Ford came to their assistance both as regards expense and transport. In his opinion men were taught to be scared of chloroform and Clover's bag, but he held that there was little difference between the Clover's bag method and the present method of giving ether by the open method with the mask covered with towels; the advantages were all in the favour of the former. Some men would never give anæsthetics well, while some men did so naturally. Everyone had the experience from time to time that in spite of the utmost care, smooth anæsthesia could not be obtained.

DR. F. L. GILL said that nothing had been said so far in regard to local anæsthetics. This method he regarded as ideal for the treatment of empyema. The operation could be performed without distress to the patient and there was a vast difference in the after effects. Strict instructions should be given to the nurse to lay each instrument down separately, so that there would be no clicking of instruments. A screen should shut off the area of operation.

Spinal anæsthesia was indicated for amputations through the hip joint in which operation there was always a very considerable degree of shock. This method was also very successful for prostatectomy. The method was to inject one cubic centimetre of a 5% "Stovaine" solution in the sitting posture and then after five to ten minutes to allow the patient to lie down. A nerve block of tenth, eleventh and twelfth nerves was also done.

In regard to intratracheal anæsthesia, there was a good deal of discussion as to whether it was required for operations on the upper part of the abdomen. In the speaker's experience the absence of diaphragmatic movements made it the anæsthetic of choice for operations on the gall bladder.

DR. A. WEBSTER said that the onlooker saw most of the game. It was just forty years since he had seen Sir William Macewan instructing in anæsthetics and he had seen almost every man in the room giving anæsthetics and he had seen a death every year for thirty-three years. The method he had been taught was the corner of the handkerchief kept moist with chloroform and the anæsthetist's ear to the patient's mouth. Of the thirty-three deaths he had seen thirty had been due to chloroform.

DR. F. J. CLARK said that it was hard to dogmatize on the question of anæsthetics. In regard to Clover's bag there was often a great deal of heavy and jerky movement which was not nice for operative work on the gall bladder. He preferred the intratracheal anæsthesia with which there was an almost complete absence of diaphragmatic movement. Occasionally after intratracheal anæsthesia there was severe vomiting, but very often the anæsthetist was not to blame, the cause being the manipulation by the surgeon. The washing out of the anæsthetic from the lungs by air helped considerably in diminishing the vomiting and after effects. It was advantageous for operations with the patients in awkward positions. Dr. Clark said that intratracheal anæsthesia was the best method. He also was of the opinion that the patients vomited less after operations in the lower portion of the abdomen if this method were used.

Referring to gas and oxygen, Dr. Clark said that when operation had been refused for chronic empyema because of the anæsthetic risk, abdominal section could be carried out under local anæsthesia and the gall bladder could be drained under gas and oxygen anæsthesia. He always used "Codrenine" for the skin, "Novocain" for the deeper tissues and quinine and urea for the peritoneum as the local anæsthetics for abdominal section.

Gas and ethyl chloride mixture was very suitable for small operations and also for empyemata. Dr. Clark agreed with Dr. F. Gill that spinal anæsthesia was indicated in operations on the aged and also for amputation through the hip joint.

The speaker always used "Stovaine" and glucose because it was rapid in its action and rapidly fixed; the patient could be placed in the recumbent position after five minutes. Dr. Clark gave it as his opinion that headaches would not be met after spinal injections provided that a sufficient amount of fluid was withdrawn prior to the injection.

DR. J. DAY said that during the summer months 90% of the anæsthetics in the Perth Hospital were given by

means of the Shipway apparatus with warm ether vapour, but during the winter months this was impossible. He said that during his term at the hospital spinal anæsthetics had been given to fifteen patients but had not proved sufficient.

Dr. F. Gill offered the opinion that the reason was that a suitable syringe was not used and that not all the anæsthetic was being introduced into the thecal canal.

Dr. Day said that he had been interested in the question of whether anæsthetics should be given to patients with auricular fibrillation and said that in his experience there did not seem to be much added risk.

DR. T. C. BOYD said that in his recent sojourn abroad he had seen this method of anæsthesia in use. There seemed to be one or two safeguards. The first was that no disinfectant be used on the back and that all the preparation was carried out with soap and distilled water. The second precaution consisted in the avoidance of preparations containing alkali. "Tropococaine" was being used as it had the same specific gravity as the cerebrospinal fluid. In London he had seen light ether anæsthesia used in conjunction with this method.

DR. C. H. LEEDMAN, M.C., referring to country conditions, said that in the district in which he had been practising up till recently, use was made of the Shipway apparatus and a regional anæsthesia of the abdominal wall was induced in all patients. As a result there was less than half the amount of after vomiting that he had seen since in the city. Dr. Leedman was of the opinion that this method also lessened the after pain of operation.

DR. A. H. GIBSON said that the personal factor must always be present in the conduct of anæsthesia. The anæsthetist who watched his patient, no matter what the form of anæsthetic, ran less risk than the one who did not.

MEDICO-POLITICAL.

A CONFERENCE convened by the Brisbane and South Coast Hospitals Board at the request of the Queensland Branch of the British Medical Association, was held at the B.M.A. Building, Adelaide Street, Brisbane, on September 28, 1927, MR. C. E. CHUTER, representing the Hospitals Board, in the chair. The object of the conference was to discuss and consider the necessity of an intelligent survey of the needs of the community as far as hospital facilities were concerned for the present and future and to coordinate voluntary, governmental and municipal effort and control in relation to hospitals in the best interests of the sick and to prevent waste and other features detrimental to hospital development.

Those present represented the Queensland Branch of the British Medical Association, the University of Queensland, the Brisbane City Council, the Brisbane and South Coast Hospitals Board, the Mater Misericordiæ Hospital, the Ambulance Transport Brigade and certain private hospitals.

Hospital Policy.

DR. E. S. JACKSON set forth the views of the Queensland Branch of the British Medical Association on the objects of the conference. Two years before the idea of such a conference had been mooted and he understood that the present meeting was merely a step towards the holding of a bigger conference on the important matter of hospital policy and control. He then submitted a definite motion:

> That the Home Secretary be asked to appoint a technical advisory board, consisting of one representative chosen by the Home Secretary's Office, one chosen by the University, one chosen by the City Planner's Department of the City Council, one chosen by members of the medical profession and one architect chosen by the Institute of Architects, to act as a direct advisory board to the Home Secretary on all matters relating to the needs of the community as far as hospital facilities are concerned for the present and future and to select qualified architects to develope plans for such institutions, as well as to advise on other matters appertaining to the service of the sick.

He cited many examples illustrating the urgent necessity of appointing such a technical advisory committee, as far as the State of Queensland was concerned.

DR. C. E. TUCKER seconded the motion and adduced further argument in favour of the appointment of a board.

MR. C. CHUTER invited the opinion of those present.

DR. J. LOCKHART GIBSON (representing the University) emphasized the importance of the proximity of a chief base hospital to both the University and the Medical School (when built). He was eager to support the motion.

PROFESSOR E. J. GODDARD (University) said that he welcomed the conference, but that he deprecated dogmatism on the subject under discussion. He pleaded that "preconceived ideas should not gather too much momentum." He was very interested in the relation of the future medical school to the hospital. Both these entities, he thought, were matters of community interest and he urged that they should profit by the mistakes made in other parts. The medical school, he said, would be concerned with the training of medical students, with postgraduate work and with research work. These functions demanded its intimate association with and proximity to a modern, well-equipped hospital. A university had many interests other than that of a medical school, but the medical aspect of a university was a very important one. Much importance lay in the fact that Australia was a young country just making a start in this regard. The whole question of hospital policy and control was a very complex one and the object of the conference was to obtain the views and opinions of the various interests involved and of the different bodies concerned in such matters. Some day there would be some sort of a medical school established at St. Lucia (Brisbane), which was the site definitely selected by the Senate of the University.

PROFESSOR A. C. V. MELBOURNE (University) endorsed the remarks of Professor Goddard and as a member of the University Senate, he fully supported Dr. Jackson's motion.

DR. E. D. AHERN (representing the Mater Misericordiæ Hospital) was eager to lend his support also to Dr. Jackson's motion. The important things to consider were the provision of doctors and nurses, the provision of adequate funds, the organization and administration and the selection of proper people to treat. He considered that the idea behind the motion was a matter of survey rather than of statement and one of the brightest features of the whole business was that the University was so sympathetic and anxious to cooperate. He regarded the establishment of a complete first class university in Queensland a definite desideratum. He wanted to endorse Dr. Jacksons' proposition and had been instructed to say, as representative of the Mater Misericordiæ Hospital, that they would heartily support and cooperate with any scheme advanced for the help and service of the sick poor.

DR. EUSTACE RUSSELL (Brisbane and South Coast Hospitals Board and Council of the Queensland Branch) also supported the motion and spoke of the advantage of having the medical school and the medical student associated with a large hospital. Such an arrangement was most desirable, as it also gave an opportunity for research, brought out the best both in the teacher and the pupil and enabled the older men to keep in the academic atmosphere and speak with authority and conviction on medical matters. He felt there had been some doubt in the minds of previous speakers as to whether the medical school should be associated directly with the university or with the hospital. He would like to suggest a compromise: a sort of divided school of medicine, in which the purely academic subjects would be taught at St. Lucia and the more practical ones at Bowen Bridge.

DR. H. W. TILLING (Brisbane City Council) wished to state that he also was in entire agreement with the proposals both from a civic and municipal point of view.

CAPTAIN E. PIKE (representing the Ambulance Transport Brigade) did not think that he could say much about the motion under discussion, seeing that the ambulance section was not much involved in the deliberations of the conference, beyond the obvious need for cooperation at all times between the various hospitals, the Ambulance Transport

Brigade, the first aid depôts and similar organizations. He assured the proposer of the motion of his support.

MR. C. E. CHUTER referred to the limited powers of the present Hospital Advisory Board and referred to a meeting between the Home Secretary and representatives of the Queensland Branch of the British Medical Association, in the course of which Mr. Stopford had agreed to extend the scope, function and powers of the existing Advisory Board. He also mentioned some of the existing regulations concerning hospitals in Queensland and referred to the commencement of the base hospital for Brisbane at Bowen Bridge, outlining the actual work in progress at present. With regard to the proposed medical school, he thought that the large hospitals must necessarily be associated with the university and the medical school.

Professor Melbourne then desired to suggest a slight modification of the motion. He would include a provision that the committee be appointed, first to report and later on to act in an advisory capacity.

Dr. E. S. Jackson appreciated Professor Melbourne's point and hoped some slight alteration would be made. He thought that the Government should act only on the advice of the advisory board and that it should never act without it. In his previous remarks he had omitted all reference to the site of the future medical school, but he himself could see many reasons why the school should go to Victoria Park near the General Hospital and not the hospital to the university at St. Lucia. His desire was to obtain coordination and cooperation of all bodies and entities interested in the important matter of hospital policy, control and administration. It was imperative to have the medical school and the hospital in juxtaposition. He was pleased to accept Professor Melbourne's suggestion, which he would incorporate in his motion.

The motion as amended was carried without dissent.

Mr. C. Chuter explained the suggestion that had been put forward in regard to the provision of intermediate and community hospitals and stated that a beginning had been made. He pointed out that it was a difficult matter to indicate the number of beds that would be available in one, two, three, four and five years. He stated that the future plans for the provision of maternity hospitals, tuberculosis sanatoria and hospitals for the insane were in the hands of the Home Department.

DR. H. V. FOXTON (President of the Queensland Branch of the British Medical Association) moved a vote of thanks to Mr. Chuter for having presided at the meeting. This was carried by acclamation. Mr. Chuter responded.

It was resolved to convey the resolution to the Home Secretary, Mr. Stopford.

IN the report of the annual meeting of the Queensland Branch of the British Medical Association, published in THE MEDICAL JOURNAL OF AUSTRALIA, December 31, 1927, page 920, the name of Dr. S. F. McDonald was incorrectly given as Dr. A. J. McDonald, among those elected as members of the Council of the Branch.

The following is a complete list of office bearers and members of the Council for the year 1928.

President: Dr. Eustace Russell.
President-Elect: Dr. Mervyn Patterson.
Vice-President: Dr. H. V. Foxton.
Honorary Secretary: Dr. E. S. Meyers.
Honorary Treasurer: Dr. F. A. Hope Michôd.
Honorary Curator of Museum: Dr. M. Graham Sutton.
Honorary Librarian: Dr. Neville G. Sutton.
Representatives on the Federal Committee: Dr. W. N. Robertson, C.B.E., and Dr. E. Sandford Jackson.
Assistant Honorary Secretary: Dr. L. J. Nye.
Members of Council: Dr. D. G. Croll, C.B.E., Dr. C. M. Lilley, Dr. S. F. McDonald, Dr. A. P. Murphy, Dr. Val McDowall, Dr. H. W. Tilling and Dr. D. E. Trumpy.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Lane, Raymond Charles, M.B., Ch.M., 1914 (Univ. Sydney), Castlereagh *viâ* Penrith.
Mulhearn, Norman St. Clair, M.B., Ch.M., 1921 (Univ. Sydney), Bellingen.
Pritchard, Denis Adrian, M.B., Ch.M., 1923 (Univ. Sydney), H.M.A.S. *Moresby*, G.P.O., Sydney.
Stevenson, Robert Baynton Comrie, M.B., Ch.M., 1924 (Univ. Sydney), Government Savings Bank, North Sydney.

Medical Societies.

MEDICAL DEFENCE SOCIETY OF QUEENSLAND.

THE ANNUAL MEETING OF THE MEDICAL DEFENCE SOCIETY OF QUEENSLAND was held at the B.M.A. Building, Adelaide Street, Brisbane, on December 13, 1927, DR. J. ESPIE DODS, D.S.O., M.C., the President, in the chair.

Annual Report of Council.

The annual report of the Council for 1927 was presented and adopted on the motion of the President, seconded by Dr. A. H. Marks.

Membership.

The Society has now a membership of 343 as compared with 316 in 1926. During the year forty-two new members were elected and one rejoined. Our loses have been as follows: twelve left the State and four deaths occurred.

The Council regrets to record the deaths of the following members: The Honourable Dr. W. F. Taylor and Dr. Andrew Stewart, both of whom were members of the Council, Dr. R. F. Bower and Dr. P. L. Townley.

Office Bearers Elected for 1928.

President: Dr. J. Espie Dods.
Vice-President: Dr. A. B. Carvosso.
Honorary Secretary: Dr. Neville G. Sutton.
Honorary Treasurer: Dr. A. H. Marks.
Auditor: Mr. R. G. Groom, F.A.C.P.A.
Members of Council:—Dr. W. N. Robertson, Dr. W. F. Taylor, Dr. Wilton Love, Dr. Kerr Scott, Dr. A. G. Anderson, Dr. D. A. Cameron, Dr. A. Stewart.

Medico-Legal.

Six cases of claims were referred to the Council by members of the Society and discussed and the advice of our solicitors was obtained. We are pleased to state that no legal action was followed in any of these cases.

Assets.

The total assets of the Society now amount to £2,442 7s. 10d.

Election of Office Bearers.

An election of three members of the Council to fill the vacancies created by the death of William Frederick Taylor and Andrew Stewart and by the retirement of Dr. Espie Dods resulted in the reelection of Dr. J. Espie Dods and the election of Dr. S. F. McDonald and Dr. R. G. Quinn.

Mr. R. G. Groom, F.A.C.P.A., was unanimously reelected Auditor for the year 1928 at the same remuneration as in previous years.

Special Correspondence.

LONDON LETTER.

By OUR SPECIAL CORRESPONDENT.

Hunterian Oration.

The first Hunterian Oration was given in 1814. This series of annual lectures was instituted as a "lasting mark of respect to the memory of the late Mr. John

Hunter" and it was arranged that it should be given in the theatre of the College on February 14, the birthday of John Hunter. The oration was to be expressive of the merits in comparative anatomy, physiology and surgery, not only of John Hunter, but also of all such persons as should be from time to time deceased, whose work had contributed to the improvement and extension of surgical science. In 1853 it was decided that the oration should be delivered biennially instead of annually.

Hunterian Lectures.

One of the clauses of the terms and conditions on which the Hunterian collection was given to the Company of Surgeons (as the Royal College of Surgeons was then known), provided "that one course of lectures, not less than twenty-four in number, on comparative anatomy and other subjects . . . ., shall be given every year by some member of the Company." In 1894 this was altered, by permission, to one course of lectures, not less than twelve in number to be given annually by a Fellow or Member of the College.

Arris and Gale Lectures.

In 1646 the Arris Lectures were founded by Edward Arris, a member of the Company of Barbers and Surgeons, of which he later became Master. The lectures were to be on anatomy. According to an Act of Parliament in 1540 the Company was entitled to claim annually four bodies of persons executed for felony and to use them for the purposes of anatomy. The surgeons were required to attend the lectures and demonstrations given on these bodies and it was for this that Edward Arris founded his lectures. When, in 1745, the union between the barbers and the surgeons was dissolved, the Arris Trust was handed over to the Company of Surgeons. The Arris and Gale bequests were the only assets which the surgeons took with them when they separated from the barber surgeons. Under the Company of Surgeons the Arris lectures were from 1753 to 1766 delivered by the Masters of Anatomy, one of the first of whom was John Hunter. In 1767 the office of Professor of Anatomy was created and the Arris Lectures were entrusted to him. The Gale Lecture was founded in 1655 by Mr. John Gale, of Bushey, a member of the Company of Barbers and Surgeons and, like the Arris Lecture, was to be on anatomy and was to be delivered every year; it was handed over to the Company of Surgeons on the dissolution of their union with the Company of Barbers. In 1810 it was decided that the Arris and Gale Lectures should be combined and it was provided that three lectures should be given annually on subjects relating to human anatomy and physiology.

Erasmus Wilson Lectures.

In 1869 Sir Erasmus Wilson founded a professorship in dermatology; he was himself appointed the first professor and from 1870 to 1878 he delivered six lectures annually. At various dates certain alterations were made in the details of the trust and it was finally decided in 1908 that a series of lectures or demonstrations should be instituted on advanced surgical pathology, illustrated by specimens in the Museum, the series comprising six lectures annually.

Bradshaw Lecture.

The Bradshaw Lecture was founded in memory of her late husband by the widow of William Wood Bradshaw, in the form of an annual lecture on surgery, to be called the "Bradshaw Lecture" and to be delivered on August 18, the anniversary of Bradshaw's death, or such other date as may be decided by the President of the College. The first lecture was given in 1882.

Thomas Vicary Lecture.

In 1919 the Company of Barbers provided a sum of money for five years for the purpose of instituting at the Royal College of Surgeons an annual historical lecture on anatomy or surgery, to be called the Thomas Vicary Lecture. This lecture is still given each year.

Arnott Demonstrations.

In 1886 Mr. J. M. Arnott (a President of the College) bequeathed a certain sum of money, to be payable on the death of his daughter. The income was to be applied annually for the purposes of the Museum and the lectures connected therewith. When the bequest was paid to the College in 1907 it was decided to institute a series of lectures or demonstrations on advanced surgical pathology, illustrated by specimens in the Museum and it is now fixed that not less than six such demonstrations be given each year by the Conservator of the Museum or other duly qualified person.

Lister Memorial Fund.

A fund was raised by public subscription in memory of Lister and in 1920 the Royal College of Surgeons became the administrators of the funds, subject to certain provisions, of which one is that every three years of a sum of £500 together with a bronze medal is awarded, irrespective of nationality, in recognition of distinguished contributions to surgical science. The recipient is required to give an address in London under the auspices of the College.

Correspondence.

THE COLLEGE OF SURGEONS OF AUSTRALASIA.

SIR: In my letters of November 5 and of December 17 on the English College of Surgeons' system of testing fitness for the Fellowship by examinations, I referred to Sir William Osler's address on "Examinations, Examiners, and Examinees," printed in *The Lancet* of October 11, 1913, page 1047, and I suggested that it would be of interest if it could be reprinted in the journal. Your note appended to my last letter shows that while this cannot be done, you can afford me space for some extracts. I gladly avail myself of the opportunity.

I have been criticized for "warmth" and for using certain descriptive expressions which I used for the same reason that Sir William Osler used them. They suited. If I was warm, Sir William Osler was warm, too—burning. No one can accuse Sir William Osler of being a careless writer. He picked and chose his adjectives, his metaphors, and his similes, and used those that were, in his opinion, best suited to the occasion. His address was a trenchantly critical review of existing medical education and medical examinations generally, but the Fellowship examination system received some of his severest criticism. But, as might have been expected from such a man as Osler, he did not find fault without suggesting a better way.

I present here some extracts, including some of the general matter because it shows the lecturer's standpoint, as well as the matter that refers more particularly to the subject at issue.

> What a student knows and what he can do—these are judged by examinations, oral, written, and practical. Tests of progress, tests on behalf of the public of fitness to follow certain callings, they have always loomed large in educational systems. At the best means to an end, at the worst the end itself, they may be the best part of an education or the worst—they may be its very essence or its ruin. Helpful if an integral part of the training, they may, and do, prove the intellectual ruin of many good men. . . . .
>
> Regarding examinations, I have one question to ask—Are they in touch with our system of education? and one suggestion to make—That from the day he enters the school, in laboratory, class-room, and wards, the work of the student should count, and count largely, in the final estimate of his fitness.
>
> Apart from a general feeling of dissatisfaction with the present system, two things strongly suggest a negative answer to the first question. As a discipline of mind and memory examinations play a leading part in all educational schemes. How they may finally control and sterilize the mind of a nation may be read in the story of China. For this has come about . . . from the blighting influence of an education directed to a single end, the passing of examinations. To test an education by its practical results at the table is to sin against the

spirit of the Greeks, who first taught the fundamental lesson that the pursuit of knowledge to be productive must be disinterested. Nothing is more fatal to a true intellectual training than a constant preoccupation with its practical results. . . . To an inquisitive mind the study of medicine may become an absorbing passion full of fascinating problems, so many of which present a deep human interest. . . . It is hard indeed to name a dry subject in the curriculum. And yet in an audience of medical students such a statement nowadays raises a smile. Why? Because we make the examination the end of education, not an accessory in its acquisition. The student is given early the impression that he is in the school to pass certain examinations, and I am afraid the society in which he moves grinds this impression into his soul. Ask *at* what he is working, and the student will answer *for* his first M.B. or his final. The atmosphere is Chinese, not Greek, and too often the one aim is to get through. We have become quite shameless about it, and practically admit a failure in our teaching when we advertise special tutorial classes for the different examinations, and consign a large proportion of our pupils to the tender care of "grinders"—and to no purpose! The spirit is taken out of instruction, and teacher and taught alike go down into the valley of Ezekiel—where they stay among the dry bones.

(Sir William Osler goes on to discuss the facts disclosed as to passes and rejections at the various final medical examinations, and towards the end considers the results attained by the candidates for the F.R.C.S., England.)

How do they fare? I am almost ashamed to read the figures. Your ears have tingled already, but only those hardened by familiarity will not be shocked at the demonstration of such a chasm between education and examination. Of 1,186 men who have tried for the primary Fellowship examination of the Royal College of Surgeons during the past five years, 821 were rejected, 69·45%. Of 680 men at the final Fellowship examination 294 were rejected, 43.23%. The high-water mark of examination futility was reached in May, 1912, when of 118 candidates for the primary Fellowship, only 31 were approved. These are picked men, our very best students, the most carefully prepared, who rarely attempt the trial without months of extra study and attendance upon grinding classes. Of the ploughed I have known personally, many seem to have been over-trained, others had spent their time in unprofitable original research; but all, passed and plucked alike, I maintain, are of the highest type of our students, whose calamities proclaim to the world the breakdown of our present educational system. . . .

Note that as far as the F.R.C.S., England, is concerned, time spent in original research is "unprofitable."

I venture to offer a few suggestions. First, by simplifying the curriculum to give the students more time. Allow the teachers a free hand in the matter of systematic lectures. Let them be reduced to a minimum or abolished altogether. One advantage they have—subjects may be dealt with which cannot possibly be illustrated in the wards. But such may better be presented in the "seminar" form, the senior students arranging the subjects among themselves under a skilled assistant. . . . I do not speak without experience when I say that the subject of medicine, for example, may be taught without the set lecture. The lecture has its value, a precious one from some lips—a Watson's or a Trousseau's; but its day has gone, to give place to other methods better adapted to modern conditions. . . .

Secondly, relief may be obtained by giving credit for work done throughout the course, changing the present system of "signing up" for one of reports by demonstrators and assistants on the character of the work done by each student. Let all who teach examine. Let education and examination go hand in hand. Let the day's work tell from the moment a student enters the school. . . . Yet, but someone will say, "Take the judgement of a group of young teachers? It is absurd." Not a bit. They see more of the students, come into closer contact, and are better able to judge of the quality of their work than the professor, and much more than any outside examiner. . . .

Thirdly, simplify the examinations. Cut off some of the written papers. In the final subjects the long report on cases, the bedside *viva*, supplemented if need be by a special "oral" will give examiners the necessary knowledge of a candidate's mental outlook. If they will consider, not how much he knows, but how he knows what he knows, the long "written" is superfluous. . . .

Fourthly, when possible, evidence of original work should be substituted for examination. Think of the stimulus to British surgery, if, in place of the Egyptian tyranny to which our best students now slavishly bow, the President and Council of the Royal College of Surgeons selected for the Fellowship each year the 15 or 20 of the men under 30 who had distinguished themselves most highly in surgical research. It would change the mental attitude of the younger generation, instil the spirit of Hunter into its members, and prevent the paralysing mental sterility that overtakes many good men who now spend precious plastic years in the dry drudgery of examination details.

Fifthly, compel no student to pass an examination twice in the same subject. . . .

The conclusion of the matter is, the student needs more time for quiet study, fewer classes, fewer lectures, and, above all, the incubus of examinations should be lifted from his soul. To replace the Chinese by the Greek spirit would enable him to seek knowledge for itself, without a thought of the end, tested and taught day by day the pupil and teacher working together on the same lines, only one a little ahead of the other. This is the ideal towards which we should move. The pity of it all is that we should have made an intolerable burden of the study of one of the most attractive of the professions, but the reform is in our own hands and should not be far off. A paragraph in an address by the late Dr. Stokes contains the pith of my remarks: "Let us emancipate the student, and give him time and opportunity for the cultivation of his mind, so that in his pupilage he shall not be a puppet in the hands of others, but rather a self-relying and reflecting being."

Sir William Osler was, beyond all dispute, the greatest teacher of our time. He has told us what he thinks about it. What are we going to do about it? Has he spoken to shut minds?

Yours, etc.,

C. E. CORLETTE.

Sydney.
December 19, 1927.

PATHOLOGICAL SERVICE.

SIR: Some time ago the Council of one of the Australian Branches issued a memorandum to members, headed "Pathological Laboratories." It was therein pointed out that a number of pathologists in private practice had asked that an inquiry be made by the Council into the question of the use of the pathological services of the Public Health Department in the case of patients financially in the position to pay a private fee.

As the result of this inquiry the Council drew the attention of members to the fact that practitioners are not justified in making use of the Government Pathologist, except in the cases of patients who cannot afford to pay for pathological services of the kind.

In South Australia, Western Australia and some other States the Government laboratories undertake pathological work and a charge is made in accordance with a published scale of charges, the fee being calculated to meet the case of even the poorest class of patients; the question is not raised as to the financial position of any patient.

In New South Wales the Government laboratories undertake pathological work without making any charge whatsoever, but with the proviso that such services are only intended for those patients who cannot afford to pay the fee charged by private pathologists.

From the nature of the above mentioned memorandum it is evident that the proviso is more honoured in the

breach than the observance. And that being so, it is also evident that there is some misunderstanding or some lack of understanding between the pathologists and the practitioners. Either the practitioner looks upon the pathologist as a highly-paid specialist, whose fees are such as only the patient in better circumstances can afford, or the pathologists have failed to let the practitioner know that rather than compel the practitioner to seek the assistance of Government laboratories, where no charge is made at all, they are prepared to give their services at a reduced fee in such cases where the usual fee cannot be afforded.

The question of pathological service is not a personal matter between the practitioner and the pathologist. It is a serious matter and concerns the administration of public health. And if difficulties are put in the way and if disputable questions arise in connexion with the carrying out of laboratory work, then there is something radically wrong.

If the practitioner is of the opinion that the usual pathologist's fees are beyond the means of the average patient, then either agitation should be got up in view of compelling the Government to create the necessary facilities to have pathological work done as in other States for a small fee and not in the light of charity; or the pathologists should be asked to declare a policy which will obviate the necessity for such a step.

If the practitioner is compelled to act first and the Government agrees to follow in the steps of the other States or if the Federal health authorities see fit to step in, there will be small opening in the future for the practising pathologist. But whereas in the other States it is the pathologist's misfortune, in New South Wales it will be of their own doing.

Yours, etc.,

"PUBLIC HEALTH."

December 29, 1927.

WILLIAM GIBSON RESEARCH SCHOLARSHIP FOR MEDICAL WOMEN.

THE William Gibson Research Scholarship for Medical Women, endowed in perpetuity by the gift of £6,000 from Miss Maud Margaret Gibson as a memorial of her father, the late Mr. William Gibson, of Melbourne, is awarded by the Council of the Royal Society of Medicine on the advice of a special committee as provided by the trust deed. The income of the fund (after payment of expenses), approximately £290, is awarded for a period of two years (with possible renewal for a third year) to a qualified medical woman upon the nomination of the scholarship committee without examination or thesis.

Dr. Alice Bloomfield has been granted an extension of the scholarship for a third year. The next award will therefore be made in June, 1929.

COMMERCIAL FLYING IN THE UNITED STATES.

IN a recent issue of the *United States Daily* it was announced that the organization of the medical service of the aeronautic branch of the Department of Commerce under Dr. L. H. Bauer, Medical Director for Aeronautics, was nearing completion.

The information is of particular interest as it is a recognition of the principle enunciated by the International Convention on Air Navigation which laid down in 1921 that civil pilots must undergo rigorous examinations before being licensed. Representatives of all the larger nations took part in the deliberations of that congress and its regulations were adopted by Continental countries, England and Australia. The United States of America declined to be governed by them.

It is difficult to comprehend the official mind of the United States of America in this matter, for experience during the war proved conclusively the necessity for the thorough medical examination of pilots and special tests were devised for the purpose.

Dr. Bauer, well known as the Commandant of the Army School for Aviation Medicine at Mitchel Field and the author of "Aviation Medicine" resigned his commission to undertake the organization of the civil side of aviation medicine. With the exception of a few centres in New England, he now has medical examiners at all centres of aviation activities, in all 170 physicians, to examine pilots who seek licences under the *Air Commerce Act* of 1926.

By sad experience, the United States of America has learned and now admits that many of its aeroplane accidents were due to physical defects of its pilots. By the application of a stringent examination with particular stress on the eyes and nervous system it hopes to reduce or eliminate the accident factor. It is recognized that the greatest care must be exercised in the selection of men fit for the growing commercial service.

Naval and Military.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 133 and 137 of December 1 and 15, 1927.

AUSTRALIAN MILITARY FORCES.

First Military District.

Australian Army Medical Corps Reserve.

The age for retirement of Honorary Captain H. A. Legge-Willis is extended for a period of two years from 2nd November, 1927.

Second Military District.

Australian Army Medical Corps.

To be Lieutenant (provisionally).—Charles Robertson Cowan, 24th September, 1927.

To be Captains (provisionally).—John Colquhoun Belisario and Edmund MacArthur Sheppard, 27th October, 1927, and 8th November, 1927, respectively. *To be Lieutenant (provisionally) supernumerary to the establishment pending absorption.*—Reginald Stuart Jones, 19th October, 1927. Major W. Vickers, D.S.O., is transferred from the Unattached List, 1st November, 1927. The provisional appointment of Lieutenant G. F. Lumley is confirmed.

The provisional appointments of Lieutenants J. Steigrad and C. C. McKellar are confirmed.

Third Military District.

Australian Army Medical Corps.

Captain W. H. Godby is appointed from the Australian Army Medical Corps Reserve, 25th October, 1927. Captain H. H. Stewart is transferred (provisionally) from the Melbourne University Rifles, 4th Division, and to be supernumerary to the establishment pending absorption, 26th October, 1927.

The provisional appointment of Lieutenant D. Zacharin is confirmed. Captains H. G. Furnell and J. G. Hayden are seconded whilst attending post graduate courses abroad, 22nd April, 1927, and 9th November, 1926, respectively.

Australian Army Medical Corps Reserve.

Major R. E. Shuter is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 14th November, 1927.

Lieutenant-Colonel S. S. Argyle is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 4th December, 1927.

Fourth Military District.

Australian Army Medical Corps.

Captain R. W. Hogg, M.C., is transferred to the Australian Army Medical Corps Reserve, 11th November, 1927.

Australian Army Medical Corps Reserve.

Major W. D. K. MacGillivray is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform, 27th November, 1927. Honorary Captain J. E. Good is retired, 6th December, 1927.

Fifth Military District.

Australian Army Medical Corps.

Honorary Captain H. M. Burns is appointed from the Australian Army Medical Corps Reserve and to be Captain (provisionally) supernumerary to the establishment pending absorption, 1st November, 1927.

Australian Army Medical Corps Reserve.

To be Honorary Captain.—John Colin Ramsay Joyce, 8th November, 1927.

Books Received.

THE GLANDS OF DESTINY (A STUDY OF THE PERSONALITY), by Ivo Geikie Cobb, M.D.; 1927. London: William Heinemann (Medical Books), Limited. Crown 8vo., pp. 300. Price: 7s. 6d. net.

GROWTH OF OUR KNOWLEDGE OF HEART DISEASE, by R. O. Moon, M.A., M.D., F.R.C.P.; 1927. Longmans, Green and Company, Limited. Crown 8vo., pp. 96. Price: 3s. 6d. net.

PROBLEMS IN PSYCHOPATHOLOGY, by T. W. Mitchell, M.D.; 1927. London: Kegan Paul, Trench, Trubner and Company, Limited; Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 190. Price: 11s. net.

THE NEUROTIC PERSONALITY, by R. G. Gordon, M.D., D.Sc., F.R.C.P. (Edinburgh); 1927. London: Kegan Paul, Trench, Trubner and Company, Limited. Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 310. Price: 12s. 6d. net.

HANDBOOK OF DISEASES OF THE NOSE, THROAT AND EAR, by W. S. Syme, M.D., F.R.F.P. *et* S.G., F.R.S.E.; Second Edition; 1927. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 415, with illustrations. Price: 12s. 6d. net.

PHYSICAL DIAGNOSIS, by Richard C. Cabot, M.D.; Ninth Edition; 1927. London: Baillière, Tindall and Cox. Royal 8vo., pp. 558, with illustrations. Price: 25s. net.

Diary for the Month.

JAN. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 27.—Queensland Branch, B.M.A.: Council.
FEB. 1.—Victorian Branch, B.M.A.: Branch.
FEB. 3.—Queensland Branch, B.M.A.: Branch.
FEB. 7.—Tasmanian Branch, B.M.A.: Council.
FEB. 9.—Victorian Branch, B.M.A.: Council.
FEB. 9.—Central Southern Medical Association, New South Wales.
FEB. 10.—Queensland Branch, B.M.A.: Council.
FEB. 14.—Tasmanian Branch, B.M.A.: Branch.
FEB. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 20.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments.

Dr. N. Ross Smith (B.M.A.) has been appointed a Medical Referee for Orthopædic Cases to the Workers' Compensation Commission of New South Wales.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

THE ADELAIDE CHILDREN'S HOSPITAL, INCORPORATED: Three Resident Medical Officers.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD: Resident Medical Officer.

THE UNIVERSITY OF MELBOURNE: Director of Bacteriological Laboratory.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 66, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £1 for Australia and £1 5s. abroad *per annum* payable in advance.